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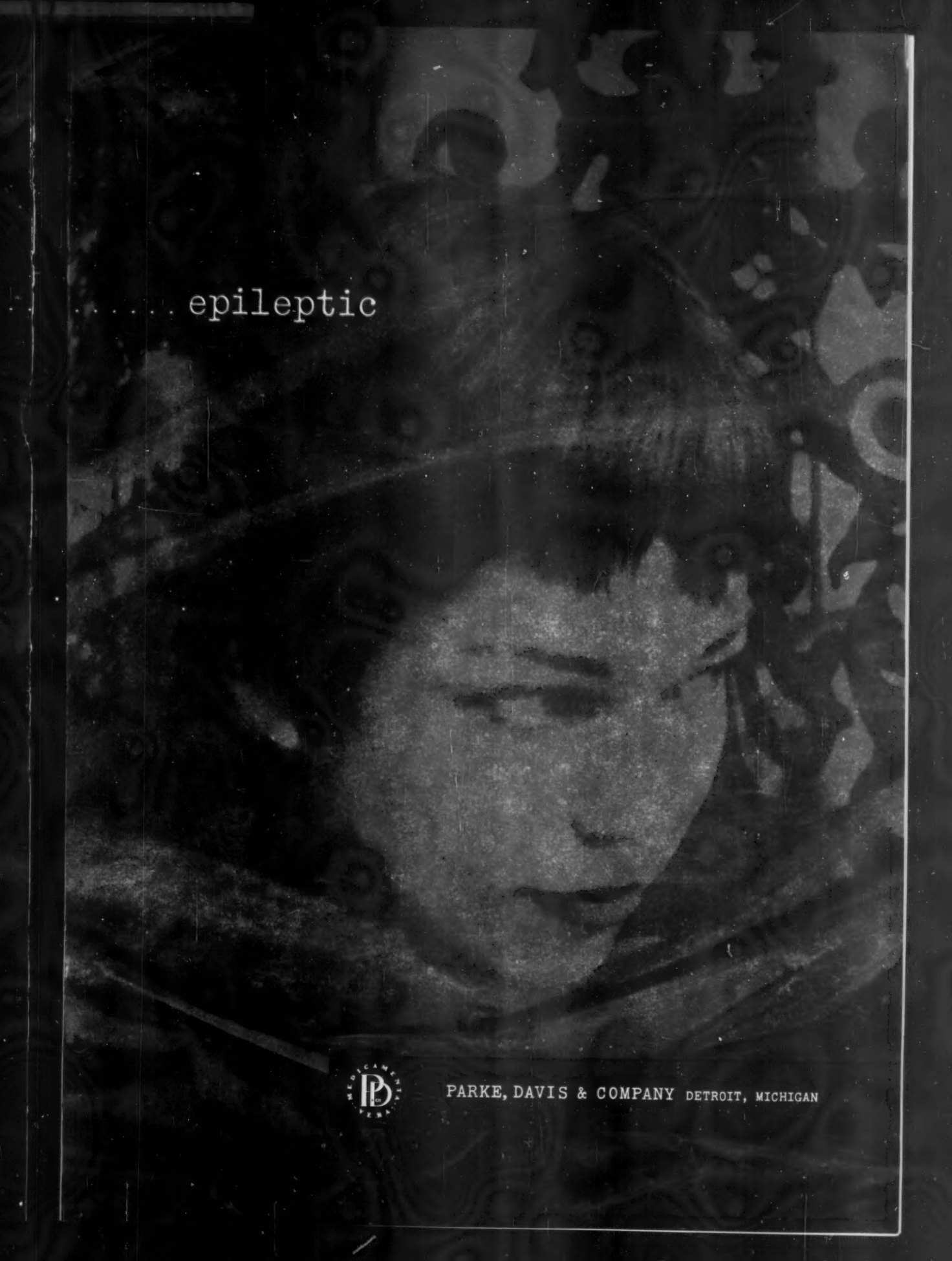

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(Founded by Landon B. Edwards, M. D., April 1874)

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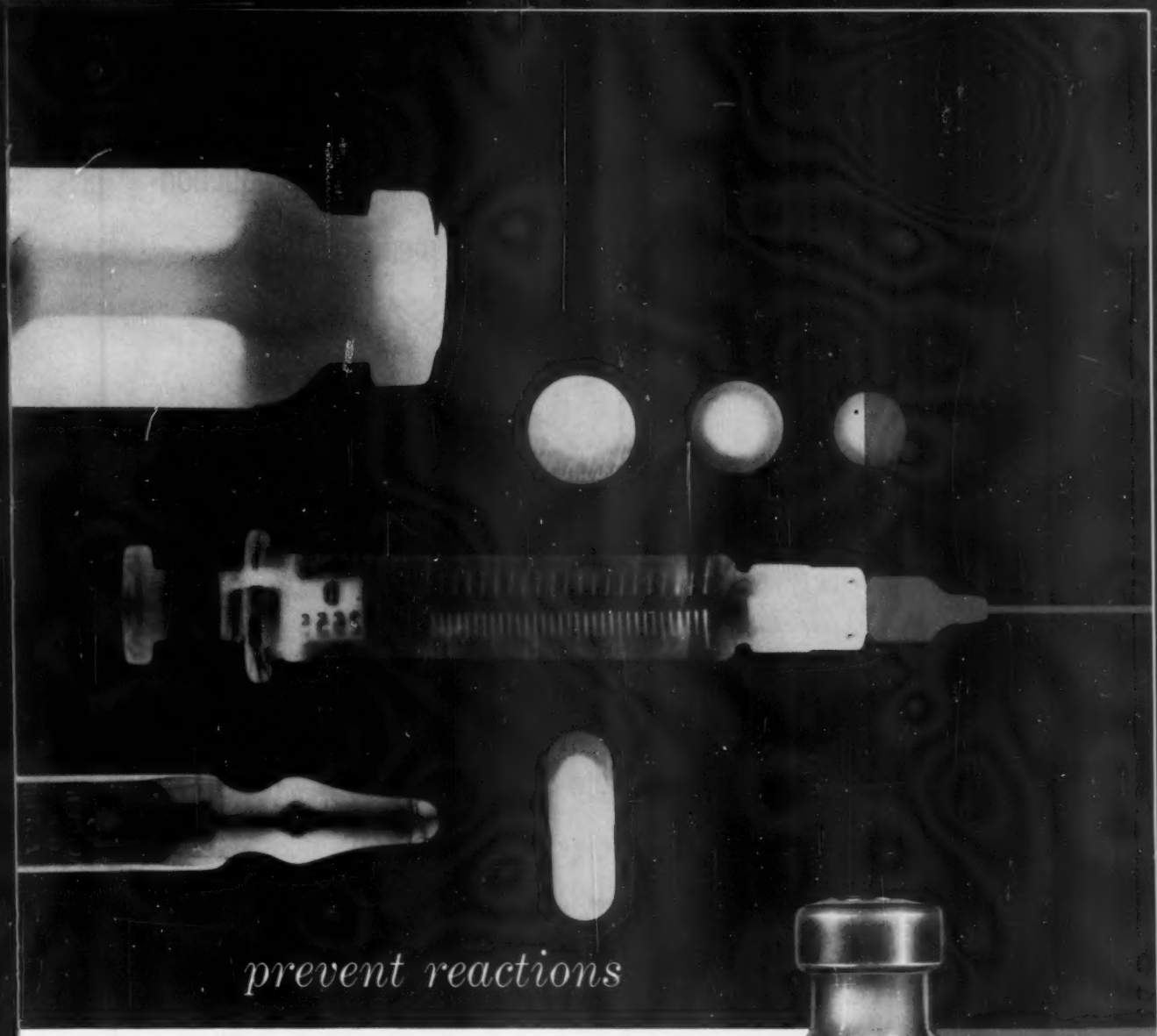

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By enrichment according to official regulations, white bread became an important food for supplying thiamine, riboflavin, niacin, and iron to the national dietary.[2] Supplementing enrichment in these nutrients, nonfat milk solids are added in amounts averaging 4 per cent (by weight) of the flour component.[3] Such enriched bread is valuable not only for its contained B vitamins and iron, but for its calcium[4] and its good quality protein[5] as well.

Mortality and morbidity of nutritional deficiency diseases have dropped markedly since the advent of commercial enriched bread. No stronger evidence can be cited than the virtual elimination of pellagra in our population in recent years.[1] In 1937, pellagra mortality was 2.5 per 100,000. By 1951 it had dropped to 0.1, representing an unprecedented low of 208 deaths reported in the entire country.

But a fall in mortality data reflects only in small measure the true improvement in public health resulting from the nutritional betterment of the national dietary.[1] Of greater concern is the vast number of people who, as a result, enjoy better health with increased capacity to work and enjoy life.

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The nutritional statements made in this advertisement have been reviewed and found consistent with current medical opinion by the Council on Foods and Nutrition of the American Medical Association.

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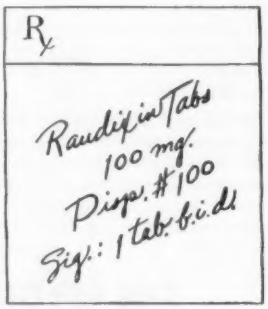

*Ataractic, from ataraxia: calmness untroubled by mental or emotional excitation. (Use of term suggested by Dr. Howard Fabing at a recent meeting of the American Psychiatric Association.)

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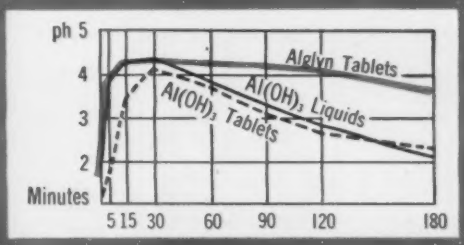

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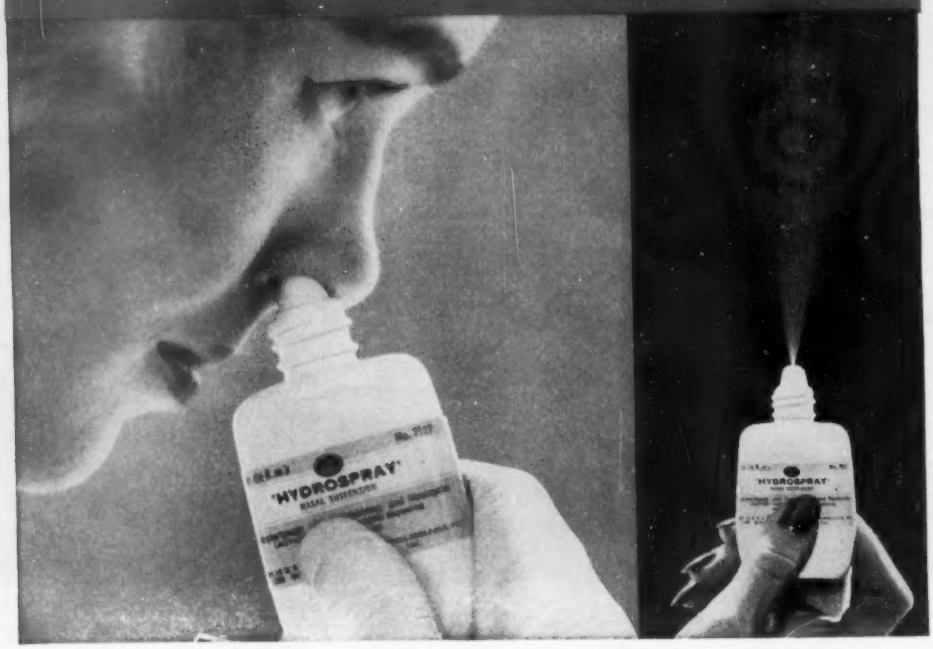

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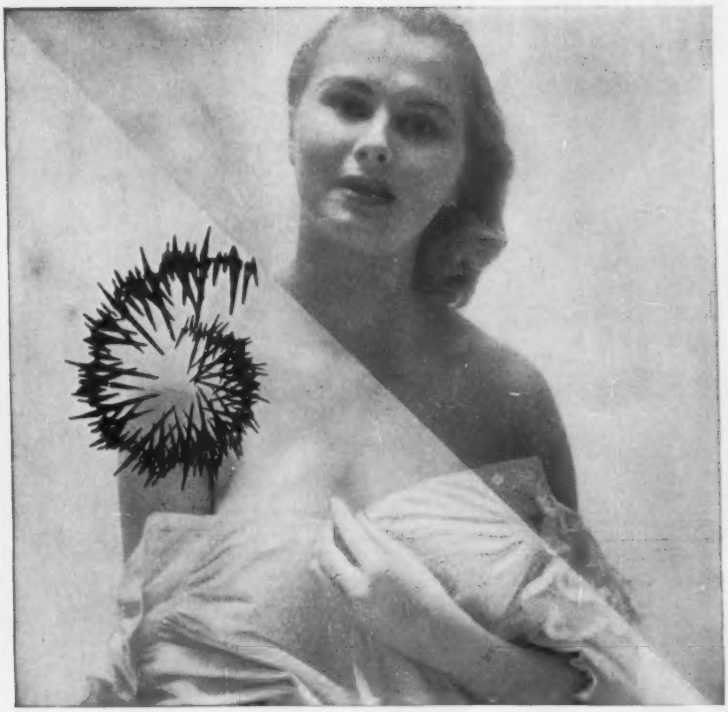

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1. Howell, T.H.; Harth, J.A.P. and Dietrich, M.: Practitioner *173*:172.
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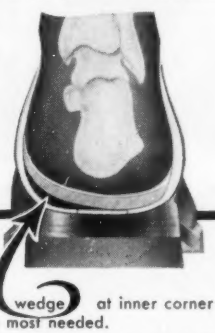

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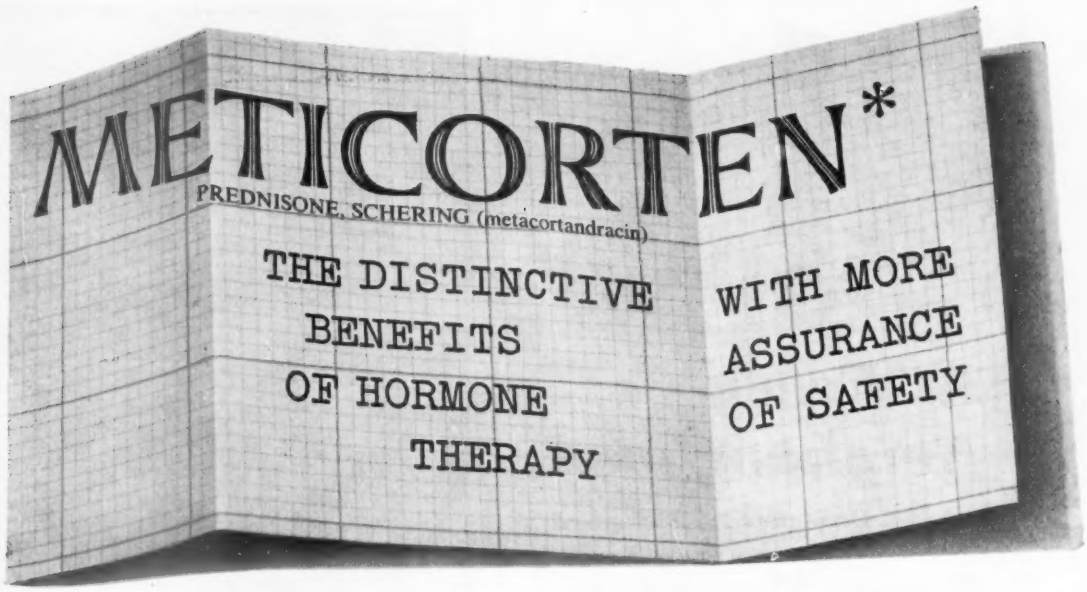

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BIBLIOGRAPHY

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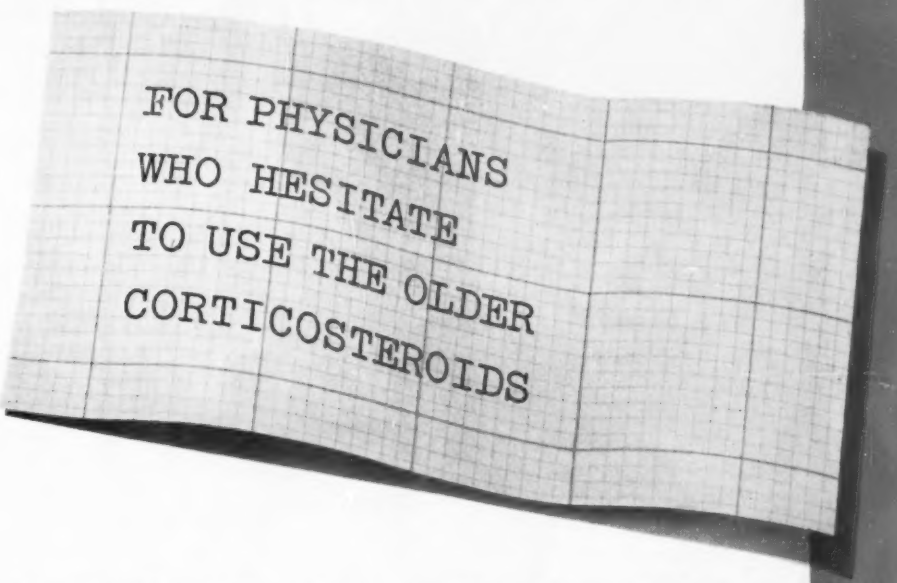

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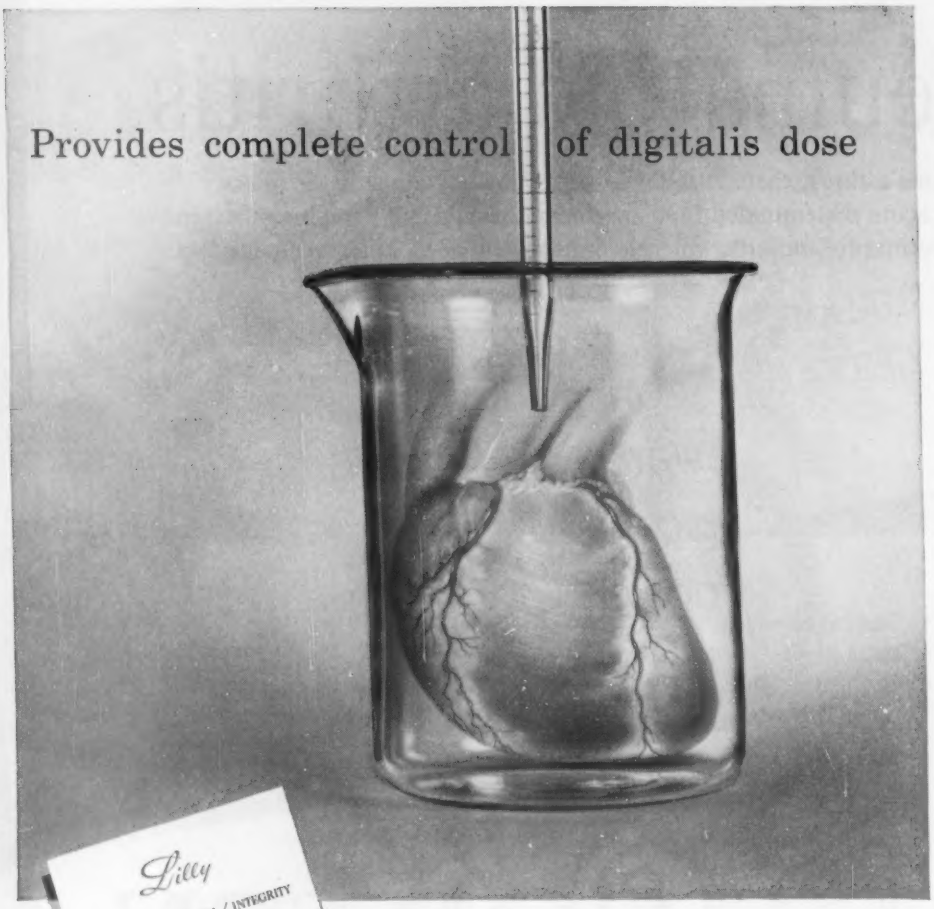

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Guest Editorial . . . .

Private and Public Medical Practice

MANY times since the close of World War II we have heard and read that organized medicine is undergoing transition and that its basic concepts are changing. We have also heard of the difficulties experienced by those who hope to predict the future with exactitude. Perhaps a more accurate statement of the situation is that organized medicine is merely keeping abreast of the changing social conditions that are occurring in every part of the world.

Since the turn of the century, progress in the conquest of disease has improved the general health of the population. As a result, man's longevity has been increased beyond our expectations. Dr. John Lynch, commenting on the remarkable longevity of Americans, states in his erudite paper, "Geriatrics Unlimited", presented to the Richmond Academy of Medicine in 1950 and published in the Virginia Medical Monthly, Volume 79, January 1952:

> "As a physician and a member of the American Medical Association, I am justly proud that this increase (in longevity) has been due in a large measure to the work of physicians and allied scientists working under a free enterprise system of government. This to me is the strongest argument against regimenting a profession which has given the world its best health and longest living."

The inter-relationships and mutual dependence of private and public practice of medicine thus becomes increasingly apparent. Fifty years ago, the extension of life through the prevention of communicable diseases was the primary objective of the practitioners of public health and preventive medicine. Efforts toward the control of communicable diseases were largely confined to such simple procedures as isolation and quarantine of the infected and the restriction of the activity of those persons in the population who had been exposed to contagion. Community health programs were designed to protect the public through the safe-guarding of public water and food supplies and the proper disposal of sewage. Thus, early public health work was restricted to disease prevention programs, and health agencies assumed responsibility for providing medical care to only the indigent in the population.

From these minor beginnings in medical care, the responsibility for providing medical care has continually grown. In the last fifty years, the improved nutritional status of the population, better housing, and improved environmental conditions have made radical changes in the structure of our society. These advances in the general level of living coupled with even greater advances in the practice of medicine have increased man's longevity by 48 years. Thus, public health is today faced with problems centered around the aged and chronically ill in the population. Today's public health administrator is concerned with the provision of structured medical services for these groups.

The provision of these services, i.e., outpatient diagnostic clinics and detections centers for the ambulant patient, adequate hospital beds for the community's acute and long-term sick, rehabilitation and retraining services, nursing homes and convalescent facilities presents a challenge to both the health worker and to organized medicine.

Increasingly, there is a demand on the part of industry for total services. No longer is industry concerned only with pre-employment examinations, first-aid services, accident prevention, plant safety programs, and the like. Both industry and labor are demanding complete and comprehensive health and medical services. Public interest in mental illness is focusing attention on preventive mental hygiene and is creating a demand for more realistic and basic prevention programs. Modern technological advances are causing changes in our basic sanitation programs. Environmental sanitation is rapidly undergoing modernization. It becomes increasingly apparent, therefore, that health agencies will have to work in closer cooperation with hospitals, with physicians in private practice, and with the variety of auxiliary medical and technological professions concerned with the population's health and well being. In brief, as medicine moves from its bio-physical era to a new bio-social phase, complete cooperation between private practitioners of medicine and public health officers becomes imperative. We believe that this cooperation can best be achieved on the local level and through joint planning wherein local Boards of Health and local medical societies work in close cooperation, reviewing every aspect of the community's health planning. Such joint planning has proven effective in the past. In the future, it will be a community's best safeguard against nationally sponsored and financed health and medical programs. Such team-work is essential if we are to safely chart the future of America's structured health programs.

The immediate past-president of The Medical Society of Virginia, Dr. Carrington Williams, in his presidential address at the 108th meeting of the society, very wisely warned the membership against the dangers of bureaucratic action, not only on the part of the federal government, but also on the part of certain voluntary agencies at their national level. We quote from Dr. Williams' address.

> "It seems to me that serious thought should also be given to the recent expansion of the activities of the American Medical Association. Its budget has multiplied many times to reach almost five million dollars for the current fiscal year. This expansion compares in proportion too closely to the federal government. Like the federal government, too, numerous bureaus have developed and expanded. The work of these groups has been uniformly good, we could not get along without them, but again from the federal government we should be warned by the power of bureaus entrenched in the security of large budgets and the same personnel whose perspective can become distorted."

Dr. Williams also commented on the increasing number of medical and scientific societies and cautioned against the increasing number of voluntary health societies and agencies. We quote:

> "The public and the doctors have organized societies on many special diseases. These societies have done much to help the victims, but they have also created false impressions of the relative importance of certain conditions."

With the trend toward national level planning on the part of both official and voluntary health and medical agencies, it is essential that organized medicine resist the tendency toward bureaucracy. We believe that this can best be accomplished through close cooperation between practitioners of public and private medicine in the towns and cities, hospitals and offices where they meet and work together.

E. M. Holmes, Jr.

Editor's Note: Dr. Holmes is Director of Public Health of the City of Richmond.

Diagnosis of Chest Pain

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Richmond, Virginia

THE correct diagnosis of chest pain is of prime importance to all physicians as this is one of the most common presenting complaints, and certainly no symptom deserves more thorough and conscientious study, for upon our diagnosis may rest the decision as to whether or not a patient may have to change his complete routine of life. The diagnosis of each case complaining of chest pain presents a grave responsibility to the conscientious physician. Harrison[1] has stated that 15-25% of patients sufficiently ill to require hospitalization complain of this symptom.

We present in this paper the data on 100 consecutive new patients seen by us in private office and hospital practice. The exact diagnosis was not clear in quite a number of these during their first consultation and at times repeated studies were necessary to establish the final correct diagnosis. Such necessary studies were carried through, and, therefore, we hope the presentation of our findings will be found of interest. In our practice, because of its specialized character, approximately 46% or new patients over the period analyzed had chest pain as their most important symptom. As a rule, these patients with chest pain were seen because they or their doctor felt that "heart pain" was present.

It is of interest, therefore, that only half of this series, which we feel is slanted heavily towards the organic by being mainly referral, had pain due to myocardial disease. Some did have pain which might be considered to be of "heart origin". These of "heart origin", but not due to myocardial ischemia, we discuss immediately below.

I. (1) Sharp, sticking precordial one-second pains: These may occur without any cardiac abnormality, but are listed here because they frequently do occur simultaneously with extrasystoles, and are abolished if the extrasystoles are abolished.

(2) Pain resembling in intensity and severity myocardial infarction, due to idiopathic pericarditis. Approximately 2% in this series have the difficulty. This type of pain may be very difficult to differentiate from myocardial infarction.

Classically, the paient will have more fever and a greater leukocytosis at the onset but this is not absolute. They will have a friction rub early, but if the history is not clear, the patient who had an almost silent infarction several days before being seen may have developed a rub by the time of first professional care. Serial electrocardiograms may or may not be of great aid in the differential diagnosis. Frequently only repeated observation will make the diagnosis clear.

The fact that the prognosis in idiopathic pericarditis is very good, whereas, the prognosis in myocardial infarction is of necessity guarded, causes the correctness of the diagnosis to be of the greatest importance.

(3) Pain resembling angina pectoris in mitral stenosis. We have had only one such patient, a white male in his 30's, who developed repeated episodes of severe squeezing substernal pain with shoulder radiation, coming on with exercise and relieved by rest, and lasting 5-10 minutes. Since his mitral valvulotomy by Dr. T. N. P. Johns, these symptoms disappeared. We feel that this is an example of the so-called "pulmonary artery syndrome" of Tinsley Harrison, which he attributes to distention of the pulmonary artery.

II. Organic non-cardiac chest pain:

(1) Hiatus hernia. Only one of the 50 chest pains carefully reviewed were due to this, which is about the true incidence, we think. In type and severity the pain may be indistinguishable from angina pectoris or myocardial infarction and may have similar radiation. Several points are of some value in substantiating a diagnosis of hiatus hernia, namely:

(a) Accentuation by meals or recumbency.

(b) Frequent presence of obesity.

(c) Absence of electrocardiographic change, and, of course, x-ray evidence of the hiatus hernia. It must be emphasized that these points are of relative value only, as the following brief history indicates.

A 39 (?) year old white male developed severe substernal pain, was seen by Dr. Richard Bowles and referred to our service. On admission he was slightly cyanotic, cold, shocky and in extreme pain. He developed a little fever and leukocytosis but five-12 lead electrocardiograms over 28 days remained normal. A gastro-intestinal x-ray series showed a small central hiatus hernia. One of the authors

Presented at annual meeting of The Medical Society of Virginia, Richmond, October 16-19, 1955.

(H. G. L.) found it difficult to believe that this represented myocardial infarction. However, when the patient returned in two months with a classical infarction he changed his mind.

Returning to the true hiatus hernia, it has been apparent in our series that many of these patients are quite emotionally upset. This may be the factor that makes an asymptomatic hernia give symptoms.

(2) Duodenal ulcer.

One patient was referred in (No. 7238) by Dr. Joseph Hoge with severe substernal pain radiating to the right shoulder, requiring morphine for relief. When the patient's symptoms subsided, the history of previous severe epigastric burning was obtained. Repeated electrocardiograms were obtained and were all normal. Gastro-intestinal x-ray series showed an active duodenal ulcer.

(3) Esophageal disease.

One patient in this series with a complaint of repeated choking and substernal burning, with a normal cardiovascular system, had a 2-3 cm. diverticulum, apparently congenital, of the mid esophagus. We think that her complaints were related to this, but it is to be recalled she was extremely nervous.

Esophagospasm and carcinoma of the esophagus, too, may be associated with pain, but we have no personal instances of this in this particular series.

(4) Disease of the spine.

A 40 year old school teacher fell into a ditch while fishing. At midnight he developed excruciating substernal pain, without radiation except slightly to the mid back. Examination disclosed an old amputation of one leg and compensatory scoliosis. There was localized tenderness over the 5th thoracic vertebrae. However, because of the excruciating pain, he was hospitalized. Electrocardiograms were normal and his back pain became more prominent. X-ray showed the same scoliosis demonstrated on physical examination.

Many diseases of the vertebrae can produce anterior chest pain; therefore, a patient with undiagnosed chest pain must have a careful and complete examination of the spine including thorough x-ray studies.

(5) Diseases of the ribs.

One of the patients had received a steering wheel blow to his chest two days previously. He complained of aching precordial pain with accentuation on breathing or exertion. X-ray of the ribs was negative but there was marked sternal tenderness.

(6) Diseases of the lungs and pleura.

The analyzed group had only two examples of this, but both pleural irritation and pulmonary embolism produce pain which may be the presenting symptom.

III. Chest pain associated with emotional difficulties and without definite organic disease.

Next to pain due to myocardial ischemia, this has been the most frequent cause of chest pain, with 44% of the chest pains falling in this group. One-half of these patients, or 22%, had an obvious precipitating cause for their complaint in the recent death of a close relative with heart disease.

It is in this group especially that a careful and detailed history and physical examination, with full work up including electrocardiograms provides a firm basis for psychotherapy.

These patients may have various tender areas on their precordium. One patient had tenderness of the costo-chondral junction (Tietze's syndrome). However, we feel that this is merely one type of the localized areas of tenderness of which these patients complain. These areas are of practical importance in that at times local infiltration with procain may give marked relief if used in conjunction with heat and handling of appropriate emotional problems.

Other minor problems may add to the chest pain. One patient's pendulous breasts seemed significant and proper support helped. Several, without any specific disease of the gastro-intestinal tract, had accentuation of their symptoms after minor dietary indiscretions, such as fried foods. We think this illustrates a general principle in these patients.

This principle is that it often requires a summation of stimuli to produce disturbing sensations. In other words, a nervous patient may not fix his anxiety unless a bruise, or pendulous breasts, or pectoral pain due to unaccustomed use of an arm provides a focus for him to center on. Then, some minor indigestion, by providing a sense of bloating and high left upper quadrant discomfort may compound his difficulty. Successful therapy requires attention to all of these factors.

We believe a careful explanation to the patient of the mechanism of his discomfort, with especial emphasis on the point that, although the discomfort may appear to him to be connected with his heart, such is not the case, will be of great help in reassuring such an anxious patient.

IV. Chest pain of myocardial origin: None of the above types of discomfort would be as important if it were not for the real necessity of correct diagnosis of pain due to myocardial ischemia.

This was present in 50% of the patients with chest pain as a presenting complaint. In three (12%) of these patients it was associated with significant

valve lesion; one with aortic stenosis, one aortic regurgitation, and one a somewhat atypical story associated with marked left ventricular enlargement and mitral regurgitation.

Character of the pain or sensation: Only 20% of the patients with pain caused by myocardial ischemia were willing to call their pain definitely squeezing, constricting or choking. 20% said it felt more like a "tightness" than the above. 12% described it as a dull ache. 9% complained primarily of burning. Some found it indescribable.

Of especial interest was the one patient that had definite accentuation of pain on breathing.

Location: All patients of this group had, no matter what their radiation, some substernal pain. This is in marked contrast to the Harrison series, where only 50% had substernal pain. However, we have encountered patients who had pain purely in the back of the neck or in the back due to myocardial ischemia.

Duration: The duration of the pain in general was, of course, more prolonged in the patients having myocardial infarction than in those having angina pectoris. We believe that pain of myocardial origin lasting over thirty minutes is rarely, if ever, due to uncomplicated angina pectoris.

Electrocardiographic findings: While we hesitate to make a definite diagnosis of myocardial infarction without electrocardiographic confirmation, it must be emphasized that this may not always occur promptly after myocardial infarction. One of these patients (No. 7248) had a normal twelve lead electrocardiogram five days after his initial pain, but when seen by us three weeks after the pain, with no further history of difficulty, there was definite electrocardiographic evidence of infarction.

Also, if the history is typical, we are quite willing to make the diagnosis of angina pectoris in the presence of a normal electrocardiogram. An exercise test may be done if there is question; however, in general, the more careful the history the less often will an electrocardiogram after exercise be necessary to establish a diagnosis. Since exercise tests in patients with angina pectoris are not without danger, we believe such tests should be used only when absolutely necessary for diagnosis. Furthermore, we believe that the electrocardiographic changes should be definite and not equivocal if the final decision concerning angina pectoris is to be based on such changes.

Summary

Many of the patients with chest pain will not have myocardial disease. Successful diagnosis is dependent upon a meticulous history and physical examination, interpreted in the light of the many factors which will cause chest pain. Severe or prolonged chest pain should be considered to be due to myocardial infarction until proved otherwise. One electrocardiogram is not adequate to rule out a myocardial infarction.

Professional Building,
Richmond Virginia

Nail Polish Sealer

A Miami physician has outdone the proverbial female who fixes everything with a hairpin or a little nail polish. Dr. Hollis F. Garrard has successfully used nail polish sealer in treating a fungal infection of the fingers and nails. He said the sealer acts as an "artificial cuticle" and keeps water and foreign material from getting under the loose skin around the nail. The sealer is applied to the nail and nail fold in the morning and left on until bedtime when it is removed and another medication applied.

The sealer, Dr. Garrard said, is superior to rubber gloves or finger cots in protecting the infected nails.

Dr. Garrard, who has used the method on 59 patients with monilial paronychia, reported the technique in the October Archives of Dermatology, published by the American Medical Association.

Penicillin Hypersensitivity

A Brief Review and Report of An Extreme Case

WM. P. COLEMAN, M.D.
OSCAR SWINEFORD, JR., M.D.
Charlottesville, Virginia

THE problem of penicillin hypersensitivity is increasing in importance, rapidly. The purpose of the following brief review, plus the report of a case of anaphylactic shock following a scratch test with penicillin, is to increase (1) the awareness of the risks of penicillin therapy and (2) the facility with which they are avoided, recognized and treated.

Case Report

A 40-year old white female was seen on 1-31-55 complaining of "Hives and Anaphylactic Shocks", intermittently for 18 months.

The urticaria occurred every three or four weeks, usually just before menstruation. She suspected milk and grains. Aspirin, previously well tolerated, now caused generalized urticaria. The penicillin history will be discussed later.

The first anaphylactic shock occurred one year previously, fifteen minutes after an injection of an estrogen preparation. Shock, unconsciousness, cyanosis, chest pain, urticaria, and difficult respiration were observed by the physician who treated the reaction. She recovered in a few hours. One month later, in a hospital, she was given an injection of demerol with no reaction. The following day, a second injection of demerol was followed promptly by a similar episode. In December, 1954, following the ingestion of an unidentified liquid medicine and some milk and crackers, she developed a mild transient reaction characterized by cyanosis, difficult respiration, and urticaria. She had avoided milk and wheat for the previous six months on the advice of her local allergist. She was then referred to this clinic.

A detailed medical survey revealed a number of problems for the relief of which the estrogen, demerol and liquid medicine had been given. Otherwise they were not related to the chief complaints.

Read before the annual meeting of The Medical Society of Virginia, in Richmond, October 16-19, 1955.

From the Allergy-Arthritis Division, Department of Internal Medicine, University of Virginia School of Medicine.

The Reaction

Many routine scratch tests were done. There was a huge reaction to penicillin. The scratch test dose of penicillin was about 500 units. After the customary waiting period of 20 minutes, intradermal tests with foods were started. Suddenly she complained of burning of skin. Several wheals were noted. She lost consciousness rapidly. Respirations slowed to four per minute. The chest was clear on auscultation. Heart sounds were very slow and barely audible. Radial pulse and blood pressure were unobtainable. She was cold, sweaty, incontinent of urine, cyanotic, and had convergent strabismus.

In rapid succession, the following therapy was started: Aqueous adrenalin subcutaneously, the shock position, 0.5 gram of aminophyllin intravenously, an intravenous drip of two ampules of norepinephrine in saline, intermittent oxygen for cyanosis, and 100 mg. of chlor-trimeton intramuscularly. Thirty minutes later, a 5% glucose infusion containing 100 mg. of hydrocortisone in alcohol was started. A strong pulse was felt in the abdominal aorta in a few minutes. A blood pressure was first detected in about forty minutes, at 80/50. It became fairly stable at 105/70 and she regained consciousness about one hour after the onset of shock. But it was seven hours before a normal blood pressure could be maintained without norepinephrine. When she became conscious, she complained bitterly of severe headache and of mid-epigastric and upper quadrant abdominal pain. An uneventful convalescence followed.

Her penicillin history as obtained initially was that she developed urticaria and/or angioneurotic edema several times after taking it during the previous eighteen months. After recovery from the shock, her family reported that she had developed (1) urticaria locally from drying her hands and arms on a towel on which some oral penicillin had been

WILLIAM P. COLEMAN, M.D., *Fellow in Allergy and Arthritis.*

spilled, and (2) generalized urticaria when she entered a room in which penicillin was being given.

Laboratory Data

An intradermal test with normal saline was negative. A scratch test with approximately one unit of crystalline penicillin was strongly positive twice. Five passive transfer sites on both arms of a volunteer were prepared with 0.1 cc. of the patient's serum. One of these sites was tested intradermally with 500 units of penicillin. All nine of the other prepared sites reacted at least as strongly as the one which was tested. This is an example of the phenomenon of contralateral passive transfer. A precipitin test with 10,000 units per cc. of penicillin was negative. The paper electrophoretic pattern of the patient's serum was not abnormal. Physical allergy tests for heat and cold were negative.

Additional tests were done, using a new syringe and needle for each dilution to avoid cross-contamination by syringes which had had stronger penicillin concentrations in them. The response to a scratch test with one one-thousandth (0.001) of one unit of crystalline penicillin was equivocal. There was no reaction to a scratch test with one ten-thousandth (0.0001) of a unit. An intradermal test using one two-hundred-thousandth (0.000005) of a unit of penicillin gave a definitely positive reaction, and the patient complained of a burning sensation in the roof of the mouth and tightness in the throat. Intradermal tests done with two one-millionths (0.000002) of a unit, and with one twenty-millionth (0.0000005) of a unit of crystalline penicillin gave equivocal reactions when compared with a saline control. Scratch tests with 1:10 and 1:00 dilutions of penicill*ium* were negative.

Titrations of the reagins in the patient's serum were positive in a dilution of 1:25, but were equivocal in the 1:125 dilution. The test sites were desensitized by the reactions. An attempt at passive local sensitization of a rabbit's skin with the patient's serum gave equivocal results. A guinea pig was not sensitized passively by her serum.

Comment

It seems reasonable to assume, in such a highly sensitive person that the previous anaphylactic reactions were due to penicillin contamination in the syringes[17]. Hyposensitization was considered too dangerous to attempt, especially since spontaneous loss of or reduction in hypersensitivity is known to occur[1,16]. Instead, she was advised to wear a metal tag warning against the use of penicillin or of syringes in which penicillin had been used. The absence of expiratory, asthma-like dyspnea during the period of shock, plus the severe abdominal pain suggests that this patient had the canine type of anaphylactic shock, in which the hepatic vein is the chief shock organ[19].

Types of Penicillin Reactions

Table I summarizes the types of reactions that may occur to penicillin. Penicillin is the commonest cause of drug allergy[1]. Fortunately most reactions are minor. The incidence of penicillin reactions of all types has been estimated at from 2 to 20 per cent[1,6,7,8]. Anaphylactic shock is the most dangerous

Table I*

Types of Penicillin Reactions

I. Hypersensitivity Reactions:
- A. General:
  1. Anaphylaxis.
  2. Herxheimer reactions, generalized.
- B. Local:
  1. Asthma
  2. Hepatitis, nephritis, hydroarthrosis, etc.
  3. Dermatoses.
     a. Contact dermatitis.
     b. Stomatitis, proctitis, urethritis, etc.
     c. Morbilliform eruptions.
     d. Vesicular or bullous reactions.
     e. Fixed drug reactions.
     f. Exfoliative dermatitis.
     g. Activation of latent fungus infections.
  4. Urticaria and/or angioneurotic edema.
  5. Reactions at injection site.
     a. Immediate inflammatory reaction.
     b. Delayed inflammatory reactions (Arthus).
     c. Granulomatous reactions.

II. Toxic Manifestations:
- A. Generalized "Toxic" reaction.
- B. Hormonal disturbances.

III. Mechanical Reactions:
- A. Nerve damage.
- B. Abscess.
- C. Intravascular administration:
  1. Vasculitis.
  2. Thrombosis or embolism.

IV. Secondary Infection:
- A. Introduction of organisms.
- B. Faulty sterilization.

V. Other Reactions:
- A. Myocarditis.
- B. L. E. Cells.
- C. Periarteritis nodosa.
- D. Superinfection.
- E. Agranulocytosis.
- F. Photosensitivity.

* Modified after Farrington.[10]

type of reaction. Hussar and Holley[2] found reports of 48 deaths from anaphylactic shock following penicillin injections, Welch *et al.*[3], in a two-year survey of 95 large general hospitals, found 59 anaphylactic reactions to penicillin. 19 were fatal. One death was due to oral penicillin. They found 25 cases of anaphylactic reactions to penathamate (Neo-Penil). Five were fatal. A history of allergic disease was present in 26 of these cases. Strauch *et al.*[4] added two anaphylactic deaths from penicillin. Eisenstadter and Hussar[5] reported a severe anaphylactic reaction to oral penicillin. Lapin[9] listed 149 reports of fatal and near-fatal immediate reactions to penicillin from January, 1953, through December, 1954. Reports of fatal and near-fatal reactions to penicillin were less frequent for children than for adults. Of the 149 cases reported by Lapin, 136 were over thirteen years of age, 61 were fatal and 75 were near-fatal. Forty-two reactions were from Neo-Penil. Undoubtedly many fatal and near-fatal reactions are not reported.

Penicillin reactions occur in many and varied forms. In addition to hypersensitivity reactions, Farrington[10] lists toxic reactions, those arising from faulty injection technic, and those involving the cardio-vascular or hematopoietic systems.

Dermatitis medicamentora may arise from oral or parenteral administration[1]. Urticaria and/or angioneurotic edema may occur promptly or as the delayed serum sickness-like reaction[10]. Erythema nodosum and erythema multiforme have been reported[8]. Contact dermatitis and mucous membrane reactions may follow the local use of penicillin[10]. Exfoliative dermatitis is a serious complication which may be fatal [11,12,13]. Vesicular or bullous dermatoses may occur alone or in association with exfoliative dermatitis[10].

The serum sickness type of reaction is common, occuring in about 3% of all patients given penicillin orally or parenterally[1,16]. The manifestations include pruritis, urticaria, joint pains, lymphadenopathy, irregular fever, and eosinophilia. Symptoms may begin 2 to 14 or more days after treatment, usually about 7 days. They usually last 5 to 10 days, but may last for 2 months or more. Purpuric reactions may occur alone or in association with other skin lesions[1,16]. Agranulocytosis[14] and photosensitivity have been reported[15,16]. A tentative role of penicillin in the etiology of disseminated lupus erythematosis and in periarteritis nodosa has been mentioned[1]. Herxheimer reactions, hepatitis, nephritis, and hydroarthrosis, may also follow penicillin[10].

Prevention of Reactions

Measures recommended to prevent or minimize penicillin reactions are: (1) Use only when definitely indicated. (2) A discussion of past penicillin experience before each series of doses. In this hospital, before the first of any series of penicillin injections is given, the nurse is required to ask if there have been any reactions to previous injections. If there has been a reaction, the nurse must have the order re-confirmed before the penicillin can be given. (3) Use oral penicillin when possible. (4) Use other antibiotics in patients who have hay fever, asthma, or atopic dermatitis.*[4] (5) Inject penicillin far enough down the extremity so a tourniquet can be applied in the event of a severe reaction[1]. (6) Do not use syringes previously used for penicillin in patients known to be sensitive to it[17].

Anaphylaxis can occur even though penicillin was well tolerated previously[16]. There is no sure way to prevent this except *not* to give penicillin. Skin tests prior to penicillin therapy are of doubtful value, since reactions may occur when skin tests are negative and penicillin may, unpredictably, be well tolerated when skin tests are positive[4,16]. Even scratch tests may be dangerous, as shown by this case.

Management of Reactions

When penicillin reactions occur, they should be treated promptly in order to lessen their severity. In the immediate severe reactions, a tourniquet should be applied proximal to the injection site if possible. The accompanying shock and cyanosis should be treated as outlined in the case report. Prompt elevation of the blood pressure is of critical importance. Wheezing should be treated with epinephrine and with intravenous aminophylline.

Immediate mild reactions, such as angioneurotic edema or urticaria, should be treated promptly with epinephrine and with antihistamine drugs. ACTH, cortisone, hydrocortisone, or, more recently, meticorten, should be used when symptoms persist in spite of repeated epinephrine and antihistamine therapy. Mild delayed reactions are treated the same way.

Severe delayed reactions which do not respond promptly to the simple therapy used for mild reactions should be treated with cortisone or its derivatives, preferably in the hospital.

There is little or no justification for the continued routine use of penicillin injections since the newer oral penicillins are highly effective, more convenient and much safer.

*This is probably no more applicable to allergic than to non-allergic patients, despite reports to the contrary.

SUMMARY

1. A severe canine type of anaphylactic reaction to a scratch test with penicillin is described.

2. A positive local and a mild systemic reaction followed an intradermal test with one two-hundred-thousandth of one unit of penicillin.

3. The types of allergic reactions to penicillin, their prevention and management are reviewed briefly.

4. Penicillin should be given by mouth instead of by injection, with rare exceptions.

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Aorta Surgery

When the aorta, the main trunk of the circulatory system, needs surgery, physicians face the problem of performing the operation without cutting off circulation. The problem is especially difficult in the arch and descending aorta, the parts lying in the chest, for "irrevocable damage" to the spinal cord may occur if the blood flow is shut off too long. However, a Boston physician, Dr. Herbert D. Adams, reported in the November 19 Journal of the American Medical Association that he successfully performed two operations on the thoracic aorta without once stopping the flow of blood. He used a method already found successful in operations on the abdominal aorta. Both operations were for the removal of aneurysms, sac-like bulges in the sides of the aortic walls.

In his operations, Dr. Adams used an aortic graft, which he inserted as a shunt or by-pass around the aneurysm. While the graft was being inserted, the blood continued to flow through the aorta. After the shunt was in place and the blood was flowing through it, the aorta was clamped shut and the aneurysm removed.

In one of the cases, involving a "huge" aneurysm, the two ends of the aorta were closed and the shunt left as the permanent passage for the blood. In the other case, a second graft was placed between the ends of the aorta as a replacement for the diseased portion and the shunt removed. In both cases the grafts functioned successfully.

Gastrojejunal Stoma

Malfunction Following Gastric Resection

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THE frequency with which gastric resections are carried out, and the fact that many of these procedures are performed by surgeons whose excursions into abdominal surgery are infrequent, justifies a report on a complication which may prove both vexatious and hazardous.

Delayed emptying of the stomach, the complication under discussion, occurs with definite frequency regardless as to the type of resection and anastomosis carried out. Glenn and Harrison,[1] in reporting one hundred gastric resections at the New York Hospital in 1950, found retention in fourteen cases, which lasted from one to thirty-one days. These patients required gastric suction for an average of 8.6 days. None required re-operation.

St. John and associates[2] found "spasm or obstruction" of the efferent loop in twelve cases and "excessive vomiting" in an additional thirty-two patients in reporting a series of three hundred and ninety-four partial gastric resections performed at Presbyterian Hospital in New York during the years 1936-1945. Allen and Welch[3] reported an incidence of 5.3% of stomal obstructions, sufficiently severe to require re-operation, following two hundred and eighty-two gastric operations. These obstructions persisted from fourteen to fifty days, following operation. Approximately the same number of patients had temporary obstruction which cleared up under conservative treatment.

These and other recent reports of temporary malfunction of the efferent loop indicate that this complication occurs in from ten to fifteen per cent of all cases of gastric resection.

This condition results from failure of the stomach to properly empty through the distal loop of the jejunum, and the symptoms usually are manifest about one week after operation. At Presbyterian Hospital in New York it frequently is referred to as "the nine day disorder."[4] The history in these cases is strikingly constant. Following a gastric resection, with associated gastro-jejunostomy, the stomach, after several days of nasal suction appears to empty satisfactorily. This situation continues until a week or ten days after operation when nausea, abdominal fullness and vomiting occur. Large amounts of fluid, including all gastric, biliary, and pancreatic secretions, as well as everything taken by mouth are vomited or withdrawn by a nasal suction tube. This condition may continue for days, weeks, or even months, in extreme cases, but ultimate disappears as mysteriously as it appeared.

Barium given by mouth during this period of obstruction will pass freely into the afferent loop of jejunum but it stops abruptly at the efferent end of the gastric stoma or penetrates only a few centimeters into the distal loop of jejunum. Various theories, none of which is too convincing, have been offered to explain this malady. Golden[4] feels that connecting the efferent loop of jejunum to the greater curvature of the stomach facilitates drainage and makes obstruction less likely. He states the erect posture causes more rapid emptying, and lying on the right side encourages drainage of an efferent loop, if placed on the lesser curvature. Once the obstruction occurs, these postural aids appear to have little influence on emptying.

Herniation of the jejunum through the defect in the transverse colon in posterior anastomoses is an easily understood but infrequent cause of obstruction.

Borden, Ravdin, and Frazier,[5] in 1937, pointed out that malfunctioning stomas later function normally and they feel that this indicates that the obstruction is not due to an improper anastomosis but that some other factor, possibly hypoproteinemia, is responsible. They state that low plasma proteins, local trauma, and large amounts of saline intraveously cause edema about anastomoses. Allen and Welch also agree with this explanation. Atony of the gastro-intestinal tract due to potassium deficits has been suggested but not proven in this type of obstruction.

Neurogenic unbalance, promoting spasm near the stoma, has been suspected in this condition but the precipitating factor, how to avoid this complication, and the long latent period before the onset of the spasm, have not been explained satisfactorily. Once this condition has developed, there fortunately is less difference of opinion as to what should be done.

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The plasma protein and potassium level should be determined at once. Lowered proteins are treated by blood transfusions and any diminution of potassium should be overcome by potassium chloride intravenously. The electrolytes must be checked at the onset and frequent determinations should be made throughout the period of obstruction. Careful intake and output records must be maintained and the returns from gastric suction, plus the twenty-four hour urinary output should provide a guide as to what solutions and how much should be given intravenously. A minimum of 3000 c.c. of 5% glucose per day will be required and at least one liter of glucose in normal saline or, better still, Ringer's

Fig. 1—Pre-operative X ray showing deformity of duodenum.

solution should be included in this total. Considerably larger amounts may be needed to offset the fluids lost by nasal suction. If the patient was in relatively good nitrogen balance at the time of operation, and if the obstruction lessens after a few days, the need for protein supplements will, in all probability, not become acute. An obstruction which persists for more than a few days, however, will require proteins intravenously or preferably by bowel. Serious consideration should be given to introducing a catheter into the upper jejunum for feeding purposes after one week of non-emptying of the stomach. If the patient is in exceptionally good condition, and relatively young, a few more days may be permitted to pass before operation is decided upon. Debilitated patients and especially older ones should be operated upon earlier.

A small amount of barium should be given by mouth prior to operation to verify the presence of a complete obstruction and to localize the site of the blockage.

A laparotomy for the purpose of placing a catheter in the upper jejunum for feeding purposes may serve a second purpose. It is usually preferable to reopen the original incision so that the gastrojejunostomy may be examined to rule out a local cause for blockage. In the case of a posterior anastomosis, the attachment of the defect in the transverse mesocolon to the stomach above the suture line should be checked to rule out the possibility of the stomach having retracted through the stoma with resulting obstruction in the adjacent jejunum. The absence of this hazard in an ante-colic anastomosis or a Billroth I resection should be considered whenever a decision is made as to the type of anastomosis which should be carried out.

Simple inspection of the anastomotic area and reassurance that the anastomosis is patent is all that should be done locally. The impulse to carry out

Fig. 2—X-ray of stomach following resection and Billroth II anastomosis showing failure of gastro-jejunostomy to function. This state of affairs continued for twenty-five days.

more drastic procedures should be resisted. The tissues are edematous, indurated, and friable. The creating of a new anastomosis usually does more harm than good. Kennedy, Reynolds, and Cantor[6] reoperated upon three patients with persisting vomiting

following gastric resection, with death in all three instances. They advise against re-operation and especially against enteroenterostomy, which was done in two of their three fatal cases.

If the indwelling nasal tube is irritating the pharynx, it also may be necessary to do a catheter gastrostomy to permit continuation of the gastric suction. This, of course, will allow removal of the indwelling Levine tube. The jejunostomy catheter for feeding purposes is inserted into the bowel about 40 cm. below the ligament of Treitz. It should be kept in mind that the condition of the patient will not permit another operation and the procedure carried out at this time must be final.

Fig. 3—X-ray evidence of rapid emptying of stomach after gastro-jejunostomy began to function.

Post-operatively the stomach contents aspirated through the nasal or gastric tube may be returned to the gastrointestinal tract through the jejunostomy and in this manner the administration of fluids and especially electrolytes is simplified. Protein and carbohydrate mixtures are given as additional feeding through the jejunostomy tube and the nutrition of the patient may be readily maintained until the jejunal loop relaxes and nourishment again may be taken by mouth.

An illustrative case is reported:

G.S.B., a forty-three year old white male, was admitted to the Retreat for the Sick on March 11, 1954, with a history of recurring bouts of epigastric pain for the past eight years. His symptoms had required hospitalization twice during the preceding three years. The disabling pain aroused him at night and had resisted the usual medical measures. Repeated gastro-intestinal X-rays showed an ulcer of the proximal duodenum without obstruction. His general physical examination and the usual laboratory tests showed nothing remarkable.

On March 12, a Billroth II partial gastrectomy, with a Hofmeister type posterior gastrojejunostomy was done. The efferent loop of jejunum was sutured to the lesser curvature of the stomach. The distal end of a nasal tube passed prior to operation was directed into the afferent loop of jejunum to relieve pressure on the closed end of the duodenum. No difficulty was encountered but 500 c.c. of whole blood was given during the procedure.

The immediate post-operative course was uneventful. The drainage through the nasal suction tube lessened and, on the fifth day, the tube was removed. The diet was increased and the stomach emptied normally. Eight days after operation and three days following removal of the nasal tube, the patient complained of gastric fullness and vomited about 700 c.c. of gastric contents. Nasal suction and intravenous fluids were resumed. Laboratory findings, including the serum proteins, were normal. Four days later a barium swallow showed no emptying of the stomach. This state of affairs continued until March 27, fifteen days following the original operation, when increasing pharyngitis, secondary to the nasal tube, necessitated a second operation.

The original incision was re-opened. A small (2 x 3 cm.) hematoma was found beneath the transverse mesocolon but no explanation was found for the failure of the anastomosis to function. A gastrostomy opening was made and a finger could be passed readily through both the afferent and efferent jejunal stomas. A large catheter with extra perforations was passed through the gastrostomy into the efferent loop of bowel to permit aspiration of the stomach and removal of the irritating nasal tube. A smaller catheter for feeding purposes was introduced into the jejunum after the Witzel technique about 40 cm. below the anastomosis.

Fluids were given by mouth and aspirated through the gastrostomy tube. This material was then inserted in the jejunostomy tube at regular intervals. Occasional blood transfusions were given and 1000 c.c. of 5% glucose in water with vitamins and frequently with potassium chloride was given daily. The upper and lower tubes were frequently connected to bridge the gap caused by the obstruction. This

did not consistently empty the stomach and it was necessary to withdraw the stomach contents at intervals and, after adding a high caloric formula, the mixture was returned to the intestinal tract through the jejunostomy tube.

This procedure was continued with minor modifications and discouraging regularity until April 14, twenty-five days after the onset of the obstruction, when, for the first time, a positive gastric balance was noted; 1,360 c.c. were taken by mouth and only 990 c.c. were withdrawn through the gastrostomy tube. Within four days the stomach emptied rapidly and completely. The patient was discharged on the thirty-fourth hospital day and has remained well.

Summary

1. Delayed emptying of the stomach after partial gastrectomy, due to malfunction of the efferent jejunal loop, occurs in ten to fifteen per cent of cases.
2. This failure of the anastomosis to function is probably due to a neurogenic unbalance, although other factors may play a part.
3. If the obstruction persists, re-operation should be performed in order that nourishment may be maintained by returning the gastric contents, plus a high protein and high calorie supplement through a jejunostomy tube. A simultaneously placed gastrostomy tube simplifies drainage of the stomach and permits the removal of the Levine tube.
4. The anastomosis should be examined to rule out mechanical obstruction but, in the absence of this, further operative procedures should be avoided. The temptation to carry out additional drastic surgical operations during this tedious period of waiting is strong, but it must be avoided and only the simplest procedure consistent with maintaining the nutrition of the patient should be done.

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Let's Reminisce!

In the Transactions of The Medical Society of Virginia for 1871, the corresponding secretary reported that in the past 12 months he had written about 25 letters, the majority of which were to the recording secretary. The number of letters received was 24. The recording secretary wrote 1100 letters during the year. "A great number of these letters never received any response—a circumstance that had added no little to the difficulties of rightly performing the duties of this office."

It was voted to pay the recording secretary an annual salary of $100.00 in addition to defraying the necessary expenses to and from the sessions of the Society.

Hepatic Coma

Clinical and Biochemical Aspects

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HISTORICALLY hepatic coma is a well-known entity and the medical writings of ancient times include descriptions of disturbed consciousness associated with jaundice by Hipprocrates and Galen. Frequent references to this phenomenon are also found in writings on liver disease during the 18th and 19th centuries. In 1860, Frerichs,[1] in an article entitled "Treatise on Liver Disease," gave one of the most complete descriptions of this neurological syndrome that has ever been made. More recently, Adams and Foley[2] have described in great detail the clinical picture of hepatic coma and correlated it with neuropathological findings in the brain. The search for the cause of this syndrome and the speculations regarding its etiology have been many in the past and have attracted the interest and the investigation of competent workers for years. Although I would not go so far as to say that the etiologic problem had been recently solved, there have been developments in the past few years that suggest that a solution is more close at hand. It is in a somewhat speculative realm that we will deal in this regard.

Originally the syndrome was attributed to a retention of bile pigments in the blood stream, and for this reason it was designated as cholemia. Frerichs[1] proved beyond a doubt that the derangement of nervous function was not related to the intensity of jaundice by showing that injections of large amounts of ox bile in the blood stream of dogs had no observable effect on the nervous system. Later it was found when more precise tests were developed that no correlation seemed to exist between an impairment of any measurable function of the liver and the neurological syndrome in question. In 1922, Matthew[3] suggested that ammonia might be a causative factor in the meat intoxication or poisoning seen in Eck fistula dogs. However, as far as amino acid excretion was concerned, there was no constant relationship between the patterns of this excretion and "hepatic coma". More recently, however, Walshe,[4] using paper chromatography, has shown that patients in "hepatic coma" may show increased amounts of glutamine along with other amino acids in the urine and plasma and that the glutamine content of the cerebro-spinal fluid may be elevated. This and other work, especially the observation of McDermott[5] and his co-workers on a patient with an Eck fistula who developed a syndrome they termed "episodic stupor" which correlated well with marked increases in blood ammonia concentration, has lead to a heightened interest in the relationship of ammonia and/or amino-acid metabolism to the syndrome referred to as "hepatic coma". But more of this relationship of protein and ammonia to hepatic coma from a clinical standpoint later. First let us examine hepatic coma itself from a descriptive point of view.

Severe liver disease is necessary for the development of hepatic coma. In general, this means decompensated liver disease which would be indicated clinically by such findings as jaundice, ascites, edema, and possibly esophageal varices and also chemically by abnormal liver function tests. Although hepatic coma may occur in individuals who are not jaundiced, this is a rather uncommon finding. Nearly all of the patients who have hepatic coma will also have ascites and some edema.[2] Even if the patient is not clinically jaundiced, it will usually be found that the serum bilirubin levels are abnormally high. The exceptions to these rules, if you will, are usually patients who have acute massive necrosis of the liver such as that as has been reported with Phenurone or carbon tetrachloride poisoning. It must also be said, however, that hepatic coma may supervene in patients who at the time may seem to be improving with regard to the signs of portal hypertension. However, in these cases we still have the triad present, in general, of jaundice, ascites and edema, despite the fact that the clinical improvement may be obvious in the sense that the ascites may be diminishing or the edema going down and the jaundice fading.

As far as precipitating factors are concerned other than that of the progressive downhill natural course of the illness itself, we might mention such things as hemorrhage from esophageal varices, intercurrent infection of almost any type. (Frequently patients with severe liver disease may develop a pneumonitis

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or pneumonia that will go unrecognized and then be followed by the onset of hepatic coma.) The use of sedative drugs is thought also in some cases to have a precipitating effect and not infrequently patients with incipient hepatic coma who are disturbed at night will be given sedatives to quiet them and in such a manner actually hasten the development of the syndrome. Finally, it has also been observed that patients having massive abdominal paracenteses will not uncommonly develop hepatic coma within a few hours after the procedure has been accomplished. Surgical procedures in a critical period of the liver disease may, of course, also precipitate hepatic coma, and this fact understandably is one of the major reasons for the rigid requirements of Linton[6] and his group in choosing patients with portal hypertension who are suitable for the spleno-renal or portocaval shunt operations.

Hepatic coma may be said to manifest itself primarily in two ways: (1) As a disturbance of consciousness, and (2) as a disturbance of motor function. Although the disturbance in motor function may be the first abnormality to be noted clinically, it is probably fair to say that, if the observer is sufficiently astute and observant, he will note that the first aberration will fall in the category of disturbed consciousness. This latter may occur either insidiously or abruptly, but is usually insidious, the first sign being that of a mild confusion. Anorexia, mental dullness, and an abnormal degree of drowsiness is often seen at the outset. Such patients may show a marked difficulty in subtracting serial sevens from one hundred or in remembering numbers with even no more than four or five digits in them. The period of confusion is usually followed by what we may call stupor, and the stupor finally progresses into coma itself. It has been reported on occasion that patients going into hepatic coma have also been quite agitated and some even have hallucinations.[2] Although the majority of the patients will show a decrease in the state or level of consciousness, there are then some patients who quite contrarily will show an agitated state rather than a depressed state of consciousness, and these patients may even verge on the maniacal. Somewhere between the time that the patient first shows signs of confusion and drowsiness and the time that they become stuporous, one is generally able to elicit a rather helpful physical sign referred to as a "flapping tremor". In this circumstance, if the patient is asked to extend the arms and to hold the extended arms before him in a rigid fashion, he will develop a flapping to and fro jerk at the hand, at the wrist, and also at the fingers, which seems to be very characteristic of patients going into hepatic coma and of those who are in hepatic coma to the extent that they are able to obey commands. However, even those who are unconscious, if the arm and hand are extended, they will sometimes demonstrate this flap. This sign may also be seen in the lower extremities when the leg is fully extended. Finally, there may be marked exaggeration of the tendon reflexes with extensor plantar reflexes and in some cases even convulsions will ensue terminally to complete the clinical picture of coma. The patient dies as if in a deep sleep. The development of the full neurological syndrome may take anywhere from a few hours to several days, and thereafter last as long as two weeks before death finally ensues. It is, however, somewhat unusual to see a patient in coma for much *more* than two weeks who might then possibly survive the episode. A certain number of patients will survive one or more episodes of hepatic coma and apparently spontaneously recover their normal state as far as their nervous systems are concerned, or at least in so far as manifestations of any disease involving the nervous system is concerned. In a considerable number of such patients the flapping tremor, if you will, may often be one of the last signs or indications of the hepatic coma and the derangement of consciousness and motor function to leave the patient. This characteristic of the stupor and even somewhat more rarely of the coma to undergo spontaneous remission, therefore, would seem to place this phenomenon of hepatic coma in the same category as the episodic stupors of hypoglycemia, acidosis, uremia, hypokalemia and Addison's disease.

Finally, we may say that the cerbro-spinal fluid in hepatic coma shows no characteristic abnormality unless the patient is intensely jaundiced, in which case the fluid may be slightly tinged by bilirubin. There is generally no increase in the number of cells in the cerebro-spinal fluid nor in the level of the protein, nor is the cerebro-spinal fluid pressure elevated. There are rather characteristic electro-encephalographic changes found in patients with hepatic coma which I shall not attempt to describe here.[7]

A syndrome that is indistinguishable from impending hepatic coma and which consists of the typical disturbance of consciousness, the flapping tremor and other neurological signs has been produced along with the typical electroencephalographic patterns in patients with advanced cirrhosis by the administration of certain nitrogenous substances.[8] McDermott and Adams[5] have produced a similar syndrome which they termed episodic stupor in a

patient with an Eck fistula by the administration of excessive amounts of protein, urea, ammonium chloride and ammonium and potassium cation exchange resin. Although knowledge of the metabolism of ammonia in humans is incomplete, it is known that an adequately functioning liver is essential in mammals for the elimination of ammonia; and here we are presented with two situations: one in which the liver is diseased and particularly incompetent as far as its functions with regard to ammonia is concerned, and, secondly, where the liver is bypassed in the case of an Eck fistula and the ammonia that is in the portal system, presumably some of it coming from urea splitting organisms in the gut itself, is re-circulated in the systemic circulation. Both cases have in common excessive amounts of circulating ammonia, although for different reasons. Although as long ago as 1922 Matthew[3] had suggested that it might be ammonia in the blood that produced the stupor in dogs with Eck fistulas given high protein diets, this entire concept has not attracted a great deal of interest until recent years when work such as that referred to re-stimulated the general interest in the relationship of proteins and ammonia to hepatic coma. It is now a rather well documented clinical fact that patients with severe liver disease who are given large amounts of protein in their diet or large amounts of ammonium chloride or other substances rich in ammonia may be expected in many cases to develop the syndrome of impending or well-developed hepatic coma. Daily doses of ammonium chloride, for example, varying from 4 grams to 14 grams per day have been seen in isolated instances to be followed by the development of the typical stupor and flapping tremor in patients with severe liver disease, only to be followed *in turn* by complete remission after elimination of the ammonia rich substance.[8] This is indeed interesting in view of the extensive use of ammonium chloride as an oral diuretic in patients with severe liver disease having also ascites and edema, and I am sure that all of us have seen cirrhotics in various stages of their illness given ammonium chloride for this reason without any observable untoward effects at all. Other patients have been observed to go into hepatic coma following a diet simply rich in protein, containing, for example, 125 grams a day of protein primarily as meat. The same story appears to be true for ammonium potassium cation exchange resins given in large doses and also for the administration of urea orally. In order to correlate, and also perhaps to explain the phenomenon of hepatic coma in patients with severe liver disease who were given substances rich in ammonia, the levels of ammonia in the blood have been measured as free ammonia, as well as nitrogenous substances in the blood capable of releasing ammonia in patients who are normal, patients who have severe liver disease, and, finally, in patients who are in hepatic coma. This is referred to as ammonia-N. Davidson's group at the Boston City Hospital, in 1953, reported the blood ammonia levels expressed as micrograms of ammonia nitrogen per millliliter of whole blood in 22 patients with severe liver disease[9] and found, comparing them with normals, the following: That normal patients, or, that is, patients without liver disease, had a mean value of 2.04 micrograms per milliliter. The patients with cirrhosis but no neurologic complications had a mean level of 2.74 micrograms per milliliter. They found a mean level in impending coma was 4.32 micrograms, and in coma to be 6. However, they also noted that individual values ranged from normal to highly elevated levels and that in serial observations the correlation between the level of blood ammonia and tremor and the state of consciousness was far from perfect. In 1954 they[10] reported ammonia studies on 10 normals, 10 patients with cirrhosis, 13 patients with cirrhosis and impending coma, and 13 patients with cirrhosis in coma. In the last paper their values were 1.3 micrograms ammonia nitrogen per milliliter for normals, 2.2 micrograms for cirrhotics without neurological complications, 2.6 micrograms for impending coma, and 3.5 micrograms for patients in coma. In the case of McDermott and Adams' patient with the Eck fistula,[5] the correlation between the periods of stupor and the levels of the blood ammonia were much better. Their range of normal for blood ammonia nitrogen was from about 29 micrograms per cent to 60 micrograms per cent and they were able to observe rises in blood ammonia to levels of 300 and 6000 micrograms per cent with the onset of stupor after the ingestion of large amounts of ammonia rich substances. Although there was an excellent correlation between the onset of stupor and the rise of the blood ammonia with the onset, the blood levels fell off rapidly after the high ammonia or high protein containing compounds were stopped. *However imperfect* the results of these and other studies may be, there is nevertheless a very suggestive correlation between the levels of blood ammonia and the onset of hepatic coma in patients with either severe liver disease or with a venous bypass of the liver who are given diets or *medications* rich in ammonia containing substances.[5-11]

Because glutamic acid is known to be oxidized by the brain[12] and may enter the Krebs cycle as alpha

keto-glutaric acid and yet may also bind ammonia to form glutamine which may be metabolized without the liberation of free ammonia, this substance

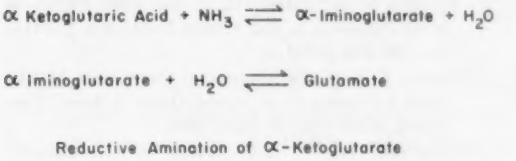
α Ketoglutaric Acid + NH3 ⇌ α-Iminoglutarate + H2O
α Iminoglutarate + H2O ⇌ Glutamate
Reductive Amination of α-Ketoglutarate
Recknagel & Potter
J.B.C. 191, 263 (1951)

Fig. 1.—Relation glutamic acid, glutamine and alpha ketoglutarate.

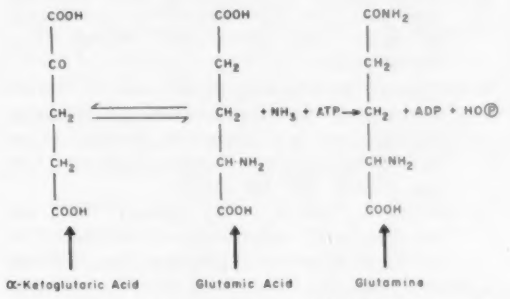

Fig. 2.—Reductive amination of alpha ketoglutarate.

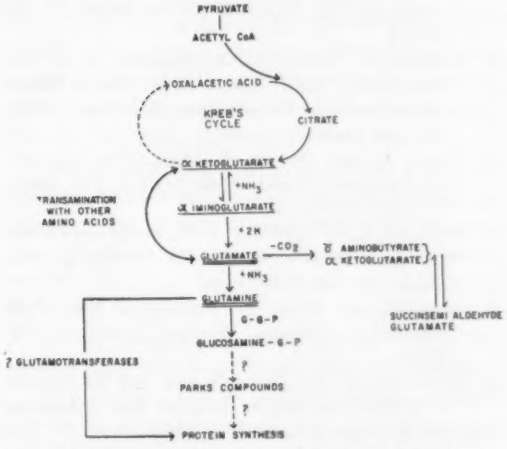

Fig. 3.—Relationship of glutamate to the Kreb's Cycle.

has been given to patients in hepatic coma with the hope of relieving them of what ammonia toxicity may be present, if an etiologic factor in the condition itself. The theory is, of course, that glutamic acid after having been ingested, would bind any of the ammonia that might be circulating in the blood in excessive amounts to form glutamine, thus preventing the theoretical reductive amination of alpha ketoglutarate[13] to take it out of the Kreb's cycle, thereby blocking or indirectly slowing up the Kreb cycle[14] and the concomitant energy production normally derived from it. Ammonia is related both to the formation and consumption of glutamic acid. Glutamic acid appears to be uniquely important in brain metabolism and it is thus possible that ammonia poisoning could be due to altered glutamic acid levels themselves. It is known, for example, that in de-proteinized extract of rat brain the four nitrogen containing compounds present in the highest concentration are glutamic acid, creatine, alphaminobutyrate and glutamine.[15] If we leave out the creatine, the remaining three account for 70% of the total free amino acids. Furthermore, these three compounds are related. It can be argued that the high concentration of glutamate indicates that it has a specific and important role in brain metabolism distinct from energy production. McIlwain has shown that glutamate fails to restore or maintain phosphocreatine in brain slices, indicating that glutamate does not ordinarily act as a fuel for energy production.[16]

The importance of glutamate may be related to its role in potassium transport in the brain as Davies and Krebs have suggested.[17] During the active transport of the potassium ion into brain cells, glutamate is taken up against a gradient mole for mole. This indicates that glutamate may carry the cation across the membrane. If this is true, a decrease in glutamate could rapidly decrease the intracellular potassium ion concentration since the exchange rate of the ion in brain cortex is much higher than in most other tissues, amounting to about 5 per cent per minute. Highly significant to our present inquiry is the finding of these workers that ammonia in the presence of an energy source such as glucose decreases the intracellular potassium. If ammonia decreases the potassium by decreasing the glutamate, this might account for the impaired brain function observed clinically in so-called ammonia poisoning that is indistinguishable clinically from hepatic coma. Thus we have at least two theories that might explain the untoward effects of excess ammonia as it is found in hepatic coma and also reasons to believe that glutamic acid might be efficacious in the treatment of hepatic coma.

It would not be fair not to mention certain drawbacks to the foregoing. The first of these is the blood brain barrier which apparently can hinder the passage of ammonia, a molecule scarcely larger than the water molecule. It has been shown that no detectable amounts of ammonia are present in cerebral spinal fluid of man despite the gradient caused by the normal blood level of about 60 micrograms per

cent.[18] The next barrier is the cell membrane which can also exert a selective influence on ammonia. It has been shown that the cerebro-spinal fluid is devoid of ammonia after electric shock convulsions which are known to raise the intracellular ammonia several-fold in the brains of rats.[12] Further barriers exist within the cell, such as the mitochondrial and presumably the microsomal membranes. It is important to remember that when we study brain slices we have removed the blood brain barrier, that hemogenates no longer have the cell membrane barrier, and that extracted enzymes lack even the barriers around the intracellular particles.

But what about glutamic acid clinically in hepatic coma. Walshe[19] has reported the administration of glutamic acid intravenously to 3 patients in 5 episodes of coma. The results were good in every case except one, which was a failure, and on one occasion the withdrawal of glutamic acid therapy appeared to precipitate coma. Other patients have been given glutamate orally and some with quite surprisingly good results. I recall a female cirrhotic who was stuporous and given 5 grams of sodium glutamate 3 times a day who within 24 hours became quite alert and who, when the medication was discontinued, again became stuporous. This procedure was repeated several times. It was finally found that she could be sent home as long as she stayed on glutamic acid and was perfectly capable of caring for herself but, as soon as the medication was stopped, would revert to a stuporous or semi-comatose condition.

In summary, I hope that I have been able to make the following points:

(1) That patients with severe liver disease who are exposed for one reason or another to large amounts of ammonia containing substances, whether they come in the diet as protein or as ammonium chloride medication, are subject to hepatic coma for this reason.

(2) That the solution to this problem, and so many other problems that we are now faced with in medicine, are to be found biochemically and that we are in an age when biochemistry is an active and necessary adjunct to the understanding and the treatment of disease processes at the clinical level, and

(3) That there is good evidence to support further investigation and use of glutamic acid or its salt glutamate in the treatment of patients with hepatic coma in conjunction with the routine therapy well-established.

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Chronic Illness in Virginia

Some Observations on the Problems

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IT IS currently estimated that there are 28,000,000 persons chronically ill in the United States; 4,000,000 are invalids. With these astonishing figures in mind, we must also consider the fact that there are more than a million persons reaching the age of 65 years of age this year and increasing each year. In more understandable figures one person in six is chronically ill and more than a thousand persons become 65 years old every day between sunrise and sunset. For emphasis, there is no prospect of any lessening of the old age load. t is little wonder that chronic illness has come to assume a major role in the problem of medical care.

A definition of chronic illness, be it physical or mental, has been defined as any illness exending beyond the acute period of thirty days. It may involve any age. In the light of the above statistics, it appears that a re-definition might be in order, or else the problem will be too big to comprehend and dim any possible solution. The chronic illness with which we are concerned is that *which requires continuing medical care in some degree beyond the usual acute period of a month's duration.* In some measure, this will reduce the problem to a workable one.

Although chronic illness is essentially a medical problem, because of its cost and the diminishing income of the patient, the individual will be compelled in many instances to seek the aid of public welfare. In fact, a large number of the chronically ill persons will look to the fields of medicine and welfare for assistance, direction and care. The two agencies must work together and share responsibility.

The many facets of the problem of chronic illness have to be evaluated in each case, i.e., as to diagnosis, prognosis, treatment and rehabilitation, with the cost of medical care usually the biggest problem. It must be borne in mind that the medical point of view to bring some degree of comfort, hope and in general to keep up the morale is the function of the family physician, or the physician who is in charge. There is no escaping the responsibility that comes with these people, who are sick.

It is the family physician who will determine what is to be done, in treatment at home—and that is the best place in the world for the chronically ill and the aged—and when he cannot longer be cared for at home, the patient must be directed by the physician to the proper place for such care as he may require. There is no agency that is in position to do this so well as the family physician. He knows the patient and he knows the patient's economic status and can advise the best plan to follow, in keeping with the needs and finances of the individual.

It is the generally accepted medical thinking, and we repeat this, the chronically ill will do better at home in their own home surroundings than elsewhere. They should be kept at home in some areas with additional services of nursing aid, housekeeping aids and other assisting aids to make it possible for families with small homes, where all the adults are working, to keep the sick person in his own quarters. It is our belief that the aged and chronically ill should be kept at home in every case if possible. It will require sacrifices and patience, but the returns, in satisfaction for having done the decent things, will compensate.

Beyond this point, the facilities for the care of chronic illness may be determined by the need and the patient's finances. The level of medical service required should be explored in each case, and adapted on the basis of ability to pay. Once more the family physician is in the key position to determine what should be done.

Domiciliary care is a more pleasing name for a good boarding house, and many of the chronically ill without homes or folks to care for them find their way into such quarters. It is a very good arrangement to a certain point. It brings people of the same age group together, keeps the patient in charge of his own offairs, and keeps up his interest in things which concern him.

The nursing home implies added nursing care. In Virginia there are 150 nursing homes which care for 3,000 people. This is no small item in medical care, nor is it a small item in investment, and one that should be encouraged to become better in services. There are some prospects that these operators will set up for themselves some minimum standards,

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which will bring them into a more desirable position, and eliminate the homes that may not be up to standard. They have for many years afforded services to the chronically ill and will do so in the future, and better it is certain.

The Chronically Ill Hospital or Facility fills in a gap between the nursing home and the General Hospital. It has facilities, such as a physician in charge, internes, graduate nurses, and a close affiliation with a General Hospital, to operate on the proper level. It has some of the equipment, and renders some of the simpler services that are to be had in a General Hospital, but without the highly specialized ones, such as operating rooms, radiology and specialty equipment. A consulting staff of specialists is another feature, which may be called upon when required.

The purpose of the chronic ill facility is to offer the patient needed service at a rate compatible with his income or that of his sponsor. The public assistance cases have to be cared for within the amount allowed by the Public Welfare Boards, and the private cases on a basis as low as they might have at home. The facility looks to the day when its offering in services to internes will further prepare them to enter the field of medicine.

The General Hospital has been relied upon in the past to care for many of these cases of chronic illness, but today the cost of a hospital bed is beyond the means of many for a prolonged period. Besides, the General Hospital is dedicated to surgery, obstetrics, and short term illnesses and emergencies, and the present shortage of rooms leaves little in the way of beds for a chronic case. It appears to be a partial solution to over-crowding in the hospitals; one bed used by a patient for ninety days will take care of many for shorter periods.

This brief review of the Chronically Ill Problem leaves little doubt in any one's mind that it is in need of more emphasis. Further, it is a question of getting prepared for an over-load such as follows a catastrophe, and there certainly seems to be no prospect of lessening the load in the years ahead, if the older age groups continue to increase. It is a continuing and increasing burden on both medicine and economy.

The answers to the problem have been suggested by the studies made by the commission on Chronic Illness which was established in 1947. It was on the basis of findings in many states that the commission has suggested some solutions. Some of the suggested solutions are already in operation in this area, but other parts of Virginia are not so fortunate. The members of this society are all general practitioners, and all of us have any number of chronically ill cases with which to deal. Some of them can be kept at home, some go to nursing homes, and some to the Patrick Henry Hospital for the Chronically Ill. In some parts of the state there are no places that the chronically ill can go. Many of the senile have been placed in the mental institutions, and there they remain, for few of them do not show mental changes. The prevention of chronic illness might occupy the interest of the Public Health, since the infectious diseases are well controlled, and it will give them a permanency of occupation.

To advocate a program at this time without more information of the needs would be foolhardy. This problem can well become costly. Any movement to really solve the problem should be predicated on a a more extensive study of the whole at the state level. To this end, it seems it would be desirable to set up pilot studies for Chronic Illness, made up of the fields of medicine, Public Health and Welfare, with sufficient authority to study, to guide and to help direct localities in any effort they might initiate to provide care for the Chronically Ill.

The current General Assembly should, of course, set up funds to match the subsidies under the new amended Hill Burton Act, for Nursing Homes, Chronically Ill Hospitals, etc.

A well studied program is advocated for the state in the end to forestall the Federal Government moving into this field with its paternalistic ideas, which will be the final blow.

Hydrocortisone in Arthritis

Treatment of Case Complicated by Duodenal Ulcer

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THIS case report is that of a 72 year old white female who carries the diagnoses duodenal ulcer with symptoms of 20 years duration, psoriasis for 30 years and a so-called mixed arthritis of finger joints, wrists, elbows, shoulders, knees, ankles and back also of 20 years duration. The arthritic picture was characterized by an intermittent nature, tender enlarged joints, soft tissue swelling, limitation of motion of varying degrees (even some ankylosis). This picture, occurring in a heavy-set individual over the age of 40—a cheerful optimistic person—should, I think, be honored as a separate clinical entity, not simply as a mixed arthritis.

Mrs. L., the patient, has a 52 year old daughter, who has a similar but less severe arthritis than her mother. Both patients feared that the daughter's incapitation would be the similar to her mother's which was near invalidism.

Both patients had had excellent treatment for their arthritis. However, we will concern ourselves mostly with our 72-year old victim who had had repeated courses of salicylates, physiotherapy, vitamins, a prolonged course of gold, small doses of terramycin for about one year, and a short course of colchicine, all with discouraging results. Specific treatment for the ulcer was initiated about one year before the course of management that we will discuss, but the patient's symptoms persisted. The ulcer and the psoriasis both became worse when the arthritis was most troublesome.

Similarly, the treatment of the daughter's arthritis had been unsatisfactory. About 20 months ago we hesitantly placed the daughter on hydrocortisone, knowing, of course, that if she improved we would be confronted with the much more painful case of the mother and the relative contraindication to the use of the medication in a patient with peptic ulcer. The daughter's response was dramatic, while the complaints of the mother increased. Although there was no objective evidence of change in the arthritic process, there was definite evidence of increased activity of the ulcer. Presumably, this was the result of the patient's knowledge that hydrocortisone was contraindicated in her particular case.

At this time the patient was placed on an extremely rigid ulcer regime, partly to learn her ability to cooperate in carrying out details, and partly to give more time for study and thought. The regime, which was continued for one month, consisted of carefully selected foods alternated with antacids hourly during the waking hours, and every two hours during the night, and belladonna every six hours continuously. She was also given 60 grains of sodium salicylate (enteric coated tablets) each day. At the end of the month both the ulcer and the arthritis were practically unchanged. The patient appeared to have followed instructions very carefully.

During the same month several highly recognized physicians with special interest in arthritic disease were consulted but the use of hydrocortisone was unanimously condemned for this particular patient.

By this time it was evident that a "last ditch" situation had been reached. Because of the complex family circumstances and the disease peculiarities of this particular patient, it became necessary to reconsider a trial of hydrocortisone with full knowledge of the serious contraindications that existed.

The possible serious consequences of treating Mrs. L. with hydrocortisone were again carefully explained. The frightening threat of silent perforation was particularly emphasized but the patient and the family were very anxious to risk the dangers in the hope of some benefit to the patient.

On April 26, 1954, Mrs. L. had a complete physical examination, was weighed, had a complete blood count and urinalysis. The physical examination revealed in addition to the arthritis, a very tender area just to the right of the umbilicus and slight epigastric tenderness which was in keeping with the X-ray diagnosis of duodenal ulcer. The patient was placed on 40 mgm. of hydrocortisone per day, the ulcer regime was altered sufficiently to provide a low sodium-high potassium diet, and she was instructed to record carefully her daily fluid intake and output. She was also instructed to help to the best of her ability about the home and dairy farm.

Subjective complaints, blood pressure readings

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and examination of the joints and abdomen, weight, urinalysis and daily fluid intake and output were evaluated at the time of each visit. Hemoglobin values were obtained at frequent intervals. The patient was seen daily for two weeks and at increasing intervals thereafter.

Within 48 hours of the first dose of hydrocortisone, the patient experienced considerable relief in her joints (and was able for the first time in months to flex her fingers around the arm rest of her chair). But the ulcer picture was unchanged. After 12 days of hydrocortisone (during which time the dosage was decreased to 20 mgs. per day) the patient was without joint pain much of the time, the swelling of the fingers was greatly reduced, and mobility in all joints was moderately increased. For the first time in well over a year the patient was entirely free from ulcer symptoms, and, what was more significant, there was only slight tenderness to deep palpation over the duodenum, and this had disappeared by the end of four weeks.

For the next 14 months, the patient was maintained on daily doses of hydrocortisone, ranging from 20 to 30 mgms. depending largely on the weather. The dietary regime for the ulcer was gradually relaxed as the patient improved, but four meals per day were maintained throughout the course of hydrocortisone. Belladonna and antacids were discontinued after one year when it was felt that they were no longer necessary. During the same period the patient's ulcer history was excellent. She had a few episodes of epigastric pain which she attributed to dietary indiscretion. There was no abdominal tenderness during any of these episodes.

Toward the end of the 15 month period, the patient was taking charcoal, etc. as her only gastro-intestinal medication, charcoal tablets for gas. X-rays, prior to the hydrocortisone, one month later, and one year later, showed deformity of the duodenal bulb.

The joint picture was greatly improved at first, but exacerbations still appeared and at times the joints were very painful on less than 30 mgms. of hydrocortisone per day. (I felt that the risk of increasing the dosage was not warranted.) After 15 months of therapy, hydrocortisone was discontinued. During the course of hydrocortisone therapy there were no untoward side-effects either subjectively, on physical examination, or in the laboratory studies.

After hydrocortisone was discontinued, the arthritic complaints increased, but for two months now the patient had been fairly comfortable on Atabrine therapy. Peptic ulcer symptoms have not recurred in the 3½ months since hydrocortisone therapy was omitted.

In conclusion, I would like to re-emphasize a few points:

1. It is my opinion that the type of arthritis represented in this case should be separately classified as it does not fit the picture of either rheumatoid or hypertropic arthritis, and particularly because of the persistently optimistic attitude of the patients.
2. If a main aggravating factor of an ulcer can be eliminated, the ulcer becomes much more susceptible to a medical and dietary regime.
3. A great part of the success in the management of this case, I feel, is due to the fact that the patient was managed at home and was kept actively at work she knew was essential to the maintenance of the family.
4. A peptic ulcer need not be an absolute contraindication to the use of hydrocortisone. Like so many other medications, its risk must be evaluated in terms of the total picture of the individual patient.
5. I would like to stress again that the risk of the use of hydrocortisone in this patient was clearly understood by the patient, the family and myself.

"Remember, this is an important year for the AMEF and medical education. If each and everyone of us are going to affirm our belief in the high standards which are now taught in our medical schools; if you and I are going to stand up for our beliefs, then you and I must sit down and give."

—Elmer Hess, M.D., *President*
American Medical Association

The Motor Innervation of the Urinary Bladder in Animals and Man

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Sympathetic Nerve Supply

THE sympathetic nerve supply to the bladder is derived from the lower thoracic and upper lumbar segments of the spinal cord. Pre-ganglionic fibers traverse the ventral spinal roots and white communicating rami to terminate in lumbar and sacral sympathetic trunk ganglia or associated plexuses of the abdomen and pelvis. Post-ganglionic sympathetic neurons have their cells of origin in the lumbar and sacral sympathetic trunk ganglia, and in the ganglia associated with the celiac, aortic, inferior mesenteric, hypogastric, and pelvic plexuses.

The abdominal aortic plexus is situated between the superior and inferior mesenteric plexuses. It is continuous above with the celiac and superior mesenteric plexuses and below with the inferior mesenteric and hypogastric plexuses. In the cat[14] the abdominal aortic plexus consists of two strands of fibers which descend along the lateral surfaces of the aorta and communicate with one another across its ventral aspect. This plexus may receive the lumber splanchnic nerves which consist of three or four strands arising from the third to the sixth lumbar segments of the sympathetic trunks[40]. Kuntz and Moseley[22] found that the lumbar splanchnic nerves in the cat do not join the aortic plexus but enter either the inferior mesenteric or hypogastric plexus. In man the abdominal aortic plexus is often referred to as the intermesenteric plexus or nerves.[6,16,24,35] Sometimes just the inferior portion of the aortic plexus is called the intermesenteric[4,30]. The aortic plexus in man has connections similar in most respects to those in the cat. It occasionally receives the least splanchnic nerve[35] in addition to the upper two or three lumbar,[20,24] although Coates[2] believes that the lumbar splanchnics enter the inferior mesenteric plexus directly. Others,[6,30,31,32,35] however, are of the opinion that the lumbar splanchnics by-pass the inferior mesenteric plexus and communicate directly with the superior hypogastric plexus.

The inferior mesenteric plexus usually surrounds the inferior mesenteric artery, forming a ring of nervous tissue which encircles the blood vessel.[14,22,27] The inferior mesenteric plexus receives contributions directly or indirectly from the abdominal aortic plexus, the least splanchnic nerve, the lumbar splanchnic nerves, and the celiac and superior mesenteric plexuses. The inferior mesenteric plexus, in both animals and man,[14,22,41] receives the celiac roots of the celiac plexus. In the cat[14] a stout fiber bundle from the celiac plexus descends along the anterior aspect of the aorta in company with its fellow of the opposite side. In man[41] the arrangement of the celiac roots is much the same as in the cat. Frequently the roots receive contributions from the least splanchnic nerve. Occasionally the celiac roots by-pass the inferior mesenteric plexus to connect directly with the hypogastric plexus[30].

Ganglia associated with the inferior mesenteric plexus have been described for both animals and man. In the cat the ganglia are generally three[14] or four[22,27,37] in number and are located around the inferior mesenteric artery. In other animals, such as the dog, rabbit, guinea pig and monkey, a similar arrangement of the ganglia has been described[41]. There are generally one or two ganglia in the rabbit, and from one to five in the dog[12]. An accessory inferior mesenteric ganglion, located one to two centimeters below the inferior mesenteric artery, has been described[12].

In man[20,41] the inferior mesenteric ganglia are of small size, are enmeshed in the inferior mesenteric plexus, and are scattered throughout the plexus surrounding the inferior mesenteric artery[41]. Some have doubted the existence of such ganglia in man[6,30]. Coates[2] speaks of them as rudimentary, and Learmonth[30] is of the opinion that either there are no ganglia corresponding to the inferior mesenteric

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ganglia of animals, or at least the ganglia, if present, are small and are of little physiological importance.

The hypogastric plexus in both animals and man connects the abdominal aortic and inferior mesenteric plexuses with the pelvic plexus. This region of the sympathetic nervous system has a rather confused terminology. In the cat most workers[14,22,27] refer to it simply as the hypogastric nerves which are continuous superiorily with the inferior mesenteric plexus and inferiorly with the pelvic plexus. In the dog Griffiths[11] described the hypogastric as a long slender grey nerve which descends into the pelvis from the abdomen in front of the bodies of the lumbar vertebrae to join the pelvic nerves in the formation of the pelvic plexus.

In man the upper part of the hypogastric plexus has often been called the superior hypogastric plexus. According to Davis,[6] this name was first used by Sappey, who, like Haller, described it as a continuation downwards of the pre-aortic plexus. Latarjet and Bonnet[29] applied the term "pre-sacral nerve" to the superior hypogastric plexus because they believed it passed over the promontory of the sacrum and ran down over that structure[2]. Recently, one of us (L. L. J.),[16] in studying a series of dissections of cadavers, found that the superior hypogastric plexus was over the fifth lumbar vertebra and traversed the inter-iliac trigone rather than being in a more inferior position as described by Latarjet and Bonnet. Other appelations,[6] such as the great hypogastric plexus, inferior aortic plexus, aortic plexus, lumbo-aortic plexus, ilio-hypogastric plexus, as well as other terms,[9] have been given to this plexus. Hovelacque,[17] as did Delmas and Laux[7], regarded the superior hypogastric plexus as a continuation of the inter-mesenteric plexus below the inferior mesenteric artery. In addition to having direct connections with the inferior mesenteric plexus, the superior hypogastric plexus receives rami from the lumbar sympathetic ganglia as well as direct connections from the outer bundles of the abdominal aortic plexus.[16,20,24,30,31,32,35]

Some workers[8,10,35] divide the remainder of the hypogastric plexus into a middle portion, called the hypogastric nerves and an inferior division, termed the inferior hypogastric plexus. According to these workers the hypogastric nerves are formed by the bifurcation of the superior hypogastric plexus and usually consist of two or more parallel bundles of fibers. They call the fan-like expansion of the hypogastric nerves at their inferior extremities the inferior hypogastric plexus which in turn makes connections with the pelvic plexus. Other investigators[1,5,6,9,24,30] have made no clear distinction between the inferior hypogastric plexus, and the hypogastric nerves. Furthermore, they consider the inferior hypogastric plexus a direct downward continuation of the superior hypogastric plexus. Kuntz[21] avoids this problem in terminology simply by calling the entire complex the hypogastric nerves and points out that these nerves break up into numerous bundles as they extend into the pelvis to form what he calls the hypogastric plexus.

The pelvic plexus, often referred to in the older literature as the hypogastric plexus,[35,40] is formed largely by the union of the hypogastric nerves and visceral branches from the sacral nerves. The urinary bladder is supplied directly through the pelvic plexus. In man[41] the pelvic plexus is well developed and its branches are very numerous. Small ganglionic masses occur frequently, especially near the point of entry of the hypogastric nerves. The pelvic plexus is located on each side of the rectum, prostate, seminal vesicles, and the posterior part of the bladder in the male. In the female[4] the pelvic plexus has often been referred to as the utero-vaginal plexus, the cervical ganglion, or the ganglion of Frankenhäuser of which the hypogastric plexus furnishes the main sympathetic supply.

We mentioned previously that pre-ganglionic sympathetic fibers arise from the lower thoracic and upper lumbar segments of the spinal cord, traverse the ventral roots, spinal nerves and white communicating rami to enter the sympathetic trunks and their ganglia. Many of the pre-ganglionic fibers terminate here. Harris[14] has shown in the cat that approximately one-third of the sympathetic post-ganglionic fibers in the inferior mesenteric plexus arise from sympathetic trunk ganglia. A few post-ganglionic fibers come from the celiac or displaced celiac ganglia. These post-ganglionic fibers, together with the pre-ganglionic sympathetic fibers, enter the inferior mesenteric plexus over the lumbar splanchnic and inter-mesenteric (aortic plexus) nerves. According to Harris[14] one-fourth of the pre-ganglionic sympathetic fibers which reach the inferior mesenteric plexus make synaptic connection with inferior mesenteric ganglion cells while the remainder pass on through to terminate at lower levels. Thus the sympathetic fibers which enter the hypogastric nerves are predominately post-ganglionic but not entirely so.[14,22,26,27,36,39,40] The pre-ganglionic sympathetic fibers in the hypogastric nerve eventually terminate in relation to ganglion cells in the hypogastric nerve,[26,27,33,41] in the pelvic plexus [22,40] or in the wall of the bladder

itself[36]. Moseley[36] has found that more than forty per cent of all of the nerve cells in the wall of the bladder receive terminations from sympathetic preganglionic fibers. According to Moseley, this is contrary to what others have reported[18,38]. Kuntz and Saccommano[23] have shown that sympathetic fibers occur in all portions of the bladder wall in far greater numbers than would be required for the innervation of the blood vessels alone. On the other hand, Langworthy and Murphy[28] were unable to find any evidence of degeneration of motor terminations on the detrusor muscle following section of post-ganglionic sympathetic fibers. They concluded that parasympathetic fibers innervated the smooth muscle of the bladder exclusively.

Parasympathetic Nerve Supply

The urinary bladder receives its parasympathetic innervation by way of the pelvic roots which arise from the medial aspects of the anterior primary divisions of two or more sacral nerves. In the cat[22,25] most of the pelvic nerve fibers arise from the second and third sacral nerves with variable contributions from the first and third sacral nerves. These fibers, which number from twelve to twenty-four in the cat, join the hypogastric nerves to form the pelvic plexus[26]. In the dog[11] the pelvic is a small white nerve which arises usually by a large root from the second sacral nerve and a small root generally from the first but often from the third sacral nerve. In man Latarjet and Bonnet[29] found that both the second and third sacrals contribute fibers to the pelvic nerves. Harman[13] and Heimburger *et al.*[15] are of the opinion that the third sacral nerve contributes the dominant parasympathetic supply with some variation of the fibers from the second and fourth sacral nerves. The latter authors found contributions more often from the second than from the fourth sacral. Occasionally the first sacral nerve in man sends fibers to the bladder[3].

The pelvic nerves usually consist of from four to ten small fibers in man but sometimes one or two larger nerves are present[35]. According to Griffiths (dog)[11] and Ashley and Anson (man),[1] the fibers of the pelvic nerves separate into three groups as they pass into the bladder wall: (1) an upper group to the fundus to the bladder; (2) a middle group to the mid-portion of the bladder; (3) a lower group of fibers to the inferior aspect of the bladder, the vesical neck and the adjacent portion of the urethra.

An accessory parasympathetic supply in the human fetus to the urinary bladder has recently been described.[19,34] It arises from the sacral nerves as they enter into the formation of the sciatic and pudendal nerves or from the pudendal nerve as it courses around the sacrospinous ligament. These nerves reach the pelvic plexus by coursing anteriorly to the inferior gluteal vessels and completely circumvent the usual parasympathetic pathway to the bladder.

Heart Attack Survey

Dr. Paul Dudley White, Boston heart specialist, has asked his colleagues to help him find out how many other Americans have had heart attacks like that of President Eisenhower. In a letter published in the November 12 Journal of the American Medical Association, Dr. White asked physicians to send him information about their own cases of acute coronary thrombosis, which is more in the limelight than it ever was before the President's illness.

He asked the doctors to supply the following:

The number of patients with unquestionable acute coronary thrombosis in their practices who became ill during the month following the President's attack —Sept. 24 to Oct. 23, 1955, inclusive, the sex, age, occupation, and national origin of each patient; and the length of time between onset of illness and any deaths.

Dr. White asked the physicians to answer even if they had had no cases during the period. He also asked them to give details about patients who became ill with coronary thrombosis before June 25, 1955.

A Note Concerning Endemic Goiter, Dental Caries and Drinking Water

FRANK L. APPERLY, M.D., F.R.C.P.
Richmond, Virginia

IT IS well known that the main causal factor in endemic goiter is an insufficient supply of iodine to the thyroid gland. This iodine poverty is found in peoples who depend for their water supply on snow water, or on water that has flowed over mountain soil or soil that has been deposited by glaciers, from which salts have long been leached. Such areas include Switzerland, New Zealand, the northern Rocky Mountain states, the region of the Great Lakes and the St. Lawrence Valley.

Much recent work seems to implicate a shortage of fluorides as one of the several causal factors in dental decay. Since we might expect fluoride poverty under the same conditions as those named above, it would not be surprising to find a higher incidence of dental decay in goitrous areas.

In spite of the numerous dental surveys made in different countries and in different areas of the United States, it is difficult to obtain comparable figures, partly because of the different criteria used. Tables I, II, and III have been constructed from figures taken from "A Survey of the Literature of Dental Caries."[1] They seem to be the only comparable figures that bear upon the problem.

TABLE I

Per cent of Carious Teeth in different countries

Locality	Age of Subjects	Per Cent of Carious Teeth
New Zealand	5 to 6	48.6%
Switzerland	5	30.6%
	6	34.3%
U. S. A.	5	13.1%

TABLE II

Per cent of children with decay experience

(Children aged 6 years)

New York State	25.3	Permanent teeth (decay, missing, filled)
Minnesota	24.3	
Maryland	12.3	
Maryland	19.9	
Md., Ga., Ill., and Mo.	20.5	
Switzerland	97	"With carious teeth"

TABLE III

Mean caries rates of soldiers from various states, stationed at a New England camp.

Area	Decayed and missing teeth per soldier
New England	12.29
Middle Atlantic	11.88
S.E. states	9.32
N.W. states	11.02
Western central states	9.19
S.W. states	8.63

These tables show a higher rate of dental caries (a) in the mountainous Switzerland and New Zealand than in the United States, and (b) in the Rocky Mountain and St. Lawrence valley states than in the other states of the United States.

Although the above facts by themselves do not prove anything, they do add something to the evidence favoring a lack of fluorides as a factor in the causation of dental caries, and they show some correlation between the incidence of endemic goiter and of dental caries.

From the Pathology Department, Medical College of Virginia.

[1] A Survey of the Literature of Dental Caries. Publication 225. National Research Council, Washington, D. C. 1952.

Received for publication September 30, 1955.

Give to the AMEF

"The very foundation of health and medical care in this country rests on our high standards of medical education. Therefore, I believe that the support of medical education should dominate our giving program".

—ELMER HESS, M.D., *President*
American Medical Association

Treatment of Glaucoma

BEN H. HINES, M.D.
Hampton, Virginia

A FIFTY-ONE year old white, married, female was first seen in my office January 19, 1954. She stated she was going to see an eye doctor in a neighboring town and she wanted a urinalysis run. She also stated she could see only a little light.

Digital examination revealed that the intra-ocular pressure was markedly increased. BP was 150/100 and urine negative.

This patient was placed on Neohydrin, one tablet daily for three days, and then one tablet weekly; Orgaphen, one teaspoonful after each meal with a little water; Optilets, two after each meal for three days, then one after each meal; and Clusivol, two daily.

The patient was seen again May 16, 1954, at which time she stated she had continued the above regime. She had noted an improved difference in one or two days and a few days later was able to see a bus for which she was waiting and at this time her vision was back to what it used to be. She further stated that prior to this treament her vision had progressively failed since October, 1953.

At this time intra-ocular pressure was not remarkable to digital examination. Also Neohydrine was increased to one daily for 5 days, then one three times weekly.

The state of improvement has been so remarkable in this one case, it would indicate further work should be done in the treatment of glaucoma with Neohydrin. Neohydrin would seem to have definite merit if used in these cases before too much damage is done due to pressure and the process is still reversible.

3804 Kecoughtan Road

Received for publication July 26, 1955.

Carbohydrate-Free Diet Impossible

Sugar in concentrated forms should be removed from the diet of a child with severe tooth decay, but other carbohydrates can't and shouldn't be completely removed. A physician consultant for the Journal of the American Medical Association was answering a physician who had questioned the advice of some dentists that "all sweets . . ., including fruits and other naturally occurring sugars," must be eliminated from the diet of caries-susceptible children.

The consultant said in the November 26 Journal that it is virtually impossible to eliminate completely all carbohydrates from the diet. He said one mother "actually was in tears" after trying to prepare such a meal. However, jams, jellies, candy, heavily sugared beverages, canned (sweetened) fruits and sweetened pastries should be avoided. The consultant also pointed out that caries-susceptible individuals should brush their teeth or at least rinse the mouth thoroughly after each meal, because decay occurs during and for about 15 minutes after eating.

Such caries activity is higher among persons who spend more time eating, either by slow eating or frequent between-meal snacks.

Heredity plays a major role in dental caries, but dietary control is still the only method of controlling the disease.

Such control has resulted in definite reduction of decay in over 80 per cent of caries-susceptible persons.

Legislative Front . . .

Social Security—Big Issue in '56

Every physician who is conscious of his duties as a citizen should now be taking an active interest in a timely issue which the American Medical Association considers of great importance—not only to the medical profession but to all of the American people.

That issue is HR 7225, a bill passed by the United States House of Representatives last summer near the end of the Congressional session. This bill, known as the Social Security Amendments of 1955, was first rushed through the House Ways and Means Committee without public hearings. Then it was passed in the House, by a vote of 372 to 31, under a suspension of the rules which barred amendments and limited debate to 40 minutes. The Senate Finance Committee, however, refused to take hasty action on a bill of such major importance. After hearing the many serious questions raised by Mrs. Hobby, then Secretary of the Department of Health, Education and Welfare, the Committee decided to hold extensive public hearings during the second session of the 84th Congress.

Just what is this legislation that appears to be so politically attractive to individuals with an eye on the 1956 elections? Why was the House majority leadership so determined to avoid open hearings and normal debate? Let's take a brief look at the main provisions of the bill.

This is the legislation which would lower the Social Security retirement age for women from 65 to 62; extend monthly benefits for permanently and totally disabled children beyond the age of 18; expand compulsory social security coverage to all self-employed professional groups except physicians, and raise social security taxes over and above the increases already scheduled for the next twenty years. Those provisions alone demand careful study of their effects on the philosophy, scope and financial stability of our social security system.

The most controversial section of the bill, however, is the one which would make permanently and totally disabled persons eligible to receive their social security retirement benefits at age 50 instead of 65. It is this section which is of particular concern to the medical profession. It is of far greater concern than the question of voluntary or compulsory coverage of physicians under the social security system. That is a separate issue which we are not discussing in this editorial. The plan for a national system of permanent and total disability benefits has far more serious implications for medicine and the nation.

It raises questions such as these: Is there any real need for a federal program? What are the facts on permanent and total disability? Won't this duplicate or overlap existing programs of assistance and rehabilitation? What effect will cash handouts have on a patient's incentive to be rehabilitated? Won't this extend federal control over physicans?—and, finally—How will this affect the future of medical practice? Will this lead, step by step, to the lowering and eventual elimination of the age-50 eligibility requirement; then, cash benefits for the dependents of those who are permanently and totally disabled; then, a temporary disability benefits program; then, cash benefits or direct government payments for hospital or medical costs, and then, ultimately, a full-fledged system of government health insurance?

These are but a few of the many grave questions which already have been raised concerning this legislation. As physicians, we must be concerned over the medical aspects of the problem. As citizens, we also must be concerned over the trends and implications in the never-ending expansion of our social security system. The minority report of the House Ways and Means Committee expressed it this way:

"We do not believe that our committee has discharged its obligation to either the Congress or to the American people by its brief and closed-door consideration of this vital legislation. We have sought to point out the grave social and economic implications of the bill. We have dwelt at some length with the staggering ultimate costs of this developing program, because we do not believe that either the Congress or the public has any conception of its magnitude."

The time has come to face up to the question of just what social security should accomplish and just where it should stop. The Association strongly *urges* that the social security issue be taken out of the arena of vote-catching politics; that there be an objective, thorough study of social security in all its present and future aspects, and that the facts and realities emerging from such a study be used as the basis for a sound national decision on this vital issue. It especially protests precipitate action on the complex question of disability without thorough investigation of alternative mechanisms.

Mental Health . . .

JOSEPH E. BARRETT, MD.
Commissioner, Department Mental Hygiene and Hospitals

Occupational Therapy for Men

In planning a program adequate to the needs of men in a state mental hospital one is impressed that over three-fourths of the prescriptions include as their objectives: an outlet for aggression, stimulation to activity, a plan for resocialization, or some opportunity for creative exploration. In attempting to facilitate these objectives in a practical work situation the therapist frequently finds that for men there is little variety in existing programs and scant provision for aggressive behavior. However, there is always a possibility for not only activity but graded participation within which the patient can be brought to recognition of his own progress. In large group activity, if the emphasis is upon the needs of the specific individual, opportunity for resocialization is present. These large group activities are frequently based upon creative expression. Therefore, if the emphasis is on the individual's need and there is a true cognizance of his place in a progressive scale, his activity can be guided to provide quite extensive creative exploration. Perhaps it would clarify the above statements if a typical large group activity were described.

Upon two year's experience in a state hospital the following evolution was noted. At Easter time in 1953 an outdoor field day event was planned with the theme centered around Easter. All games were modified to emphasize this theme. In other words, the relay races were hopping contests and in a throwing contest plaster eggs were used. Relays employed Easter baskets and colored eggs and an egg rolling contest. The plan was simple and the events were organized largely on a recall basis because all the games had been experienced. There was little participation in preparation except within the Occupational Therapy Shops where a few men were employed in laying out the field, molding the eggs and/or painting them. No participation on the planning level was provided for any patient. Posters and verbal announcements were used to stir up interest on the wards, at the weekly dances, and in the newspaper. However, although the weather was perfect and all the supplies and equipment were ready on time, half of the time was wasted in organizing the groups. The patients came in double lines and tended to remain in this formation. There was no spontaneous participation although the staff as a whole, helped by members of other staffs, worked extremely hard. Some six hundred people were on the field and participated as recipients of Easter favors. Approximately two hundred of the total group actively engaged in any games.

During the following months leading up to the July 4th celebration a different plan was initiated. The Occupational Therapy Staff met and planned the project. Various shops, according to the type and condition of their patients, underwrote definite activities. This program was centered around a rodeo. The men modified games which were housed in ten booths. The entire layout for the whole program was carried on by patients under the direction of shop therapists. The actual frames of the booths were cut and fitted in the Woodwork Shop by men. The work was assigned to each patient on the basis of experience each particular patient needed. For instance, those who needed to externalize their aggression did the sawing, the hammering and driving of stakes. It was observed that two patients with whom no rapport could be established previously, quickly responded to the responsibility of a specific job which they recognized as essential to the completion of the whole. Very sick individuals, who were slow to respond and some who were even at a smearing level of behavior, progressed from lackadaisical dabs at the framework to evident pride in a well painted and decorated booth. Groups of men, who had shown little or no interest in female patients, learned simple square dances in which they showed spontaneous enjoyment upon performance. Some initiated additional activities by making small models. These were incorporated immediately in the general program. One thousand patients attended this performance and of that group six hundred participated upon as assigned basis. This did not take into account that each patient, unless very feeble, had to collect his lunch and select an area where he could enjoy it. It entirely ignored that

Article prepared by FLORENCE E. CLEMENS, *Director of Occupational Therapy, Western State Hospital, Staunton, Virginia.*

group who, in response to the hillbilly band, danced almost the entire afternoon. The appreciation of the afternoon was expressed in a normal spontaneous manner. It is felt important that out of this type of activity the men, especially during the construction of the booths, laying out the field, building the games, decorating of the booths and assisting with the clean up, were included in a work progress which they recognized as essential and sensible.

One year later, in early preparation for the celebration of the same holiday, the schedule was planned and the theme was accepted by the Occupational Therapy Staff. The shop therapists took the idea back to their individual shops and although there had been a great deal of change in the personnel of the various groups, there was an enthusiastic response on the part of the men who volunteered almost immediate suggestions as to games, events and even the contents of their picnic supper. Perhaps it would clarify the picture of exactly how the individual needs of the men can be met in these large group activities if a case history were briefly outlined. It is readily accepted that these activities in no way solve underlying emotional problems that involve the patient but rather it prepares the climate in which he becomes accessible and can tolerate psychotherapy.

John was a 35 year old man, admitted to the hospital three months before the beginning of this program. He had had one course of thirty-five treatments of electro-convulsive therapy and was quiet enough to be taken to the shop class. He showed no interest whatsoever in taking part in any activity suggested to him. It was found that he enjoyed crossword and jigsaw puzzles. Day after day he would either solve one or assemble the other or simply sit with bowed head facing the wall. Any attempt to attract this man's attention was met by a scowl or a manneristic protrusion of the mouth. If one persisted in getting his attention, it resulted in the patient's urination in his clothes. Indirect attention and efforts to include him in any group activity met with either complete withdrawal or a scowl or a sullen flattening of his expression. In one shop, where a group of six men were discussing the general program for the 4th of July celebration, someone asked: "Are they going to have any dancing this time?". An affirmative answer was given and the therapist demonstrated one of the steps which produced spontaneous amusement. It was noted that John was watching and, responding to a remark made to him, mumbled "Yes, I can dance". An opportunity was provided for him to be included in one of the dances and although one of the costumes was rather extreme, he showed no resentment to it or to any of the practice periods. It was noted that he learned to recognize his partner and, although she was a very sick person, he took some initiative in placing her in the proper position within the circle. It was soon evident that he was an excellent dancer and he was given additional responsibility for leading the group on and off the stage. This he accepted and would even chat a little with the therapist and with some of the other men. However, most of the time his expression was one of stolid indifference unless the music was playing and the dance was actually in progress. An opportunity was created for him to express his preference as to costume color and this he could tolerate although he initiated no suggestion of his own. In the shop it was possible to approach this patient and elicit response to questions. His posture improved and he no longer found it necessary to face the wall when in the shop. Also he could assume some responsibility for both himself and for others. This was felt to be a distinct improvement over the seclusiveness he had previously demonstrated. Although the activity has long since been over, dancing is still used as a type of treatment. During one session he could accept a suggestion of the therapist that if he could explain to the doctor what was troubling him, the doctor would be anxious to help him. To date there is little carryover of the rapport established, but treatment using this medium is being continued.

The development from a more or less regimented attendance of activities planned for the people in a hospital situation to participation in games and their active contribution upon a planning level is felt to indicate progress. This is found to be encouraging but should also be accepted as a challenge to proceed to the point at which the activity will evolve from the suggestions not made by the staff but rather resulting from group interaction on the part of the men in the hospital.

Public Health . . . .

MACK I. SHANHOLTZ, M.D.
State Health Commissioner of Virginia

Virginia Program for Purchase, Distribution, and Administration of Poliomyelitis Vaccine

When the U. S. Public Health Service asked the State Health Department for its program for the purchase, distribution, and administration of poliomyelitis vaccine to be allocated to Virginia and its use of Federal funds granted under the Poliomyelitis Vaccine Assistance Act of 1955, only three requirements were stipulated to be incorporated in the program. The first of these was that there should be no means test; the second was that there should be equitable distribution throughout the state; and the third was that administration of the vaccine should be confined to the age group or groups designated at the time.

On September 14, 1955, the Poliomyelitis Advisory Committee to the State Health Department, which is the Poliomyelitis Committee of The Medical Society of Virginia, met with the State Health Commissioner to develop the Virginia program to be submitted to the Public Health Service. The executive secretary of the Virginia Pharmaceutical Association was invited to meet with the group and was given voice in all discussions. Since it was believed that Federal funds granted to Virginia would be sufficient to purchase all vaccine allocated to Virginia as it becomes available until February 14, 1956, when the grant expires, it was decided that the State Health Department would purchase all of each allotment as it is released. The representative of the druggists agreed to this because:

1. The amount of vaccine that would be available to the druggists by any other plan would be too small to handle locally.
2. There would be great difficulties in making equitable distribution.
3. There would be too much paper work for the small amount of vaccine that might be handled.
4. There would be too great danger of causing ill feeling because the amount of vaccine received by a druggist would not supply all physician customers.

The State Health Department, therefore, agreed to purchase all available vaccine as it is released. When a sufficient amount of the vaccine will have been accumulated, distribution will be made on the basis of percentage of population of each area to the designated age group or groups prevailing at the time. The distribution will be made to local health departments where one of three plans, or a combination of them, may be used in administering vaccine. It may be given in regular immunization clinics; it may be used in special, mass-immunization clinics; and it may be distributed to private physicians for use in their offices. In no case can there be a charge made for the vaccine, but physicians can be paid for their services in clinics and they can make a charge for administering the vaccine in their private offices.

It was decided that records will have to be kept and, in order to report the use of the vaccine in Virginia to the Public Health Service, the private physician should report the use of the vaccine given him to the health director of his area. The Advisory Committee further recommended that a physician may receive as much as six 9 cc. vials of vaccine at one time and must report on the use of these before being eligible to obtain additional vaccine. The physician in accepting vaccine agrees to make these reports, to confine its use to the age groups prevailing at the time of administration, and to use the vaccine before the expiration date.

The Committee advised that the public be informed through the press, radio, and television and that the private physician be informed through The Medical Society of Virginia and local medical societies. The health directors will be kept up to date through the State Health Department.

The first distribution of Salk vaccine for this program was made November first when more than 206,000 cc. were shipped to county and city health departments. The Public Health Service had just extended the age group by five years and the Advisory Committee ruled that in Virginia this should be done by adding four years below 5 and one year above 9; the priority age group is now from 1 through 10 years.

Physicians of the remaining four counties without a health department have been notified that the vaccine allotted to their areas is stored in the State Health Department and is available to them on request, as much as six 9 cc. vials at a time.

As more vaccine is released, it will be bought and stored in the State Health Department until the

amount accumulated warrants another distribution.

Recent reports made by the Surgeon-General of the Public Health Service indicate that one dose of the vaccine affords protection against paralytic poliomyelitis and that the more people given one dose now, the more protection the population will have when the next polio season is reached. These reports also indicate that the measures taken for insuring the safety of the vaccine have been successful and that the additional filtration, which has just been required, is another factor to increase our confidence in the safety of the vaccine. Through its use we can hope to reduce poliomyelitis to a disease of minimal incidence.

MONTHLY REPORT OF BUREAU OF COMMUNICABLE DISEASE

	Nov. 1955	Nov. 1954	Jan.-Nov. 1955	Jan.-Nov. 1954
Brucellosis	1	2	31	44
Diphtheria	7	5	38	33
Infectious Hepatitis	42	114	1019	3399
Measles	204	83	3999	23611
Meningococcal Infections	9	13	87	97
Poliomyelitis	11	43	322	596
Rocky Mt. Spotted Fever	1	0	46	41
Streptococcal Infections	250	263	6180	4270
Tularemia	3	1	15	32
Typhoid Fever	1	7	43	58
Rabies (In Animals)	15	29	347	340

Chemical Poisoning Treatment

Physicians have been advised on methods of treating organic phosphate poisoning, which has become increasing prevalent in recent years through the misuse of some insecticides. The potential use of these same phosphate compounds as chemical warfare agents also makes it important that physicians become better acquainted with ways of diagnosing and treating such poisoning, two University of Illinois scientists said in the November 19 Journal of the American Medical Association.

The compounds are among the most powerful insecticides yet developed and extensive agricultural use has resulted in many accidents and deaths.

The best known methods of treatment include the administration of the drug atropine sulfate to control nervous system effects; artificial respiration to treat respiratory failure, and general treatment for other symptoms.

In a review of literature on the subject, they found that atropine must be given as soon as possible and in larger than normal doses in order to combat the poisoning. The review of 25 cases showed a direct relationship between survival, the amount of atropine given, and the speed of administration. While an overdosage of atropine produces certain uncomfortable effects, they are not serious, but the consequences of inadequate treatment for organic phosphate poisoning as grave. Treatment with atropine should tend toward overdosage rather than underdosage.

The effects of the organic phosphates are prolonged and treatment must be continued "vigilantly" until all signs of poisoning are gone. Severe exposure or delayed therapy may result in death.

Other methods of treatment include: application of gas mask and/or removal from the site of the vapors; immediate washing away of any liquid contamination; drainage of excessive bronchial secretions that may block the airway; oxygen administration if necessary; treatment of the eyes with atropine to counteract eye symptoms, and administration of medicine to stop convulsions if they are not controlled by atropine.

The study by Archer S. Gordon, M.D., Ph.D., and Charles W. Frye, M.S., Chicago, of the department of clinical science, College of Medicine, University of Illinois, was supported by a grant from the Army Chemical Center Medical Laboratories, Edgewood, Md.

Current Currents

A BULLETIN from National Selective Service headquarters contains the following information concerning the Armed Forces Reserve Medical Officer Commissioning and Residency Consideration Program:

1. To insure that the Armed Forces will continue to receive medical officers who have had residency training in certain fields, the Director of Selective Service and the Department of Defense have agreed to continue the program known as the "Armed Forces Reserve Medical Officer Commissioning and Residency Consideration Program".

2. This program permits a limited number of regular registrants who are or become medical reserve officers to complete their residency training prior to being called to active duty. To implement this agreement, it is the policy of the Director of Selective Service to recommend the deferment of such registrants who may be selected by the Assistant Secretary of Defense (Health and Medical) for essential residency training under this program.

3. Selected registrants will be certified to the Selective Service System on Request for Deferment for Residency Training (SD Form 247). Upon receipt of this form properly completed, the Director of Selective Service will forward it to the registrant's local board through the appropriate State Director of Selective Service.

THE HOUSE OF DELEGATES of the American Medical Association has approved a program alerting physicians to the implications of H. R. 7225—the Bill which would make possible disability cash payments at age 50.

A report of the Board of Trustees notes that AMA's concern is with two parts of the Bill: extension of OASI benefits for totally and permanently disabled children beyond age 18, and provision of OASI retirement benefits for the disabled at age 50, instead of the present statutory requirement of age 65.

The Board declared: "This Bill clearly is another step in the extension of government over the medical profession." This is the way it would come about: By providing a disability benefit as a right through the OASI system, it would subject doctors to government regulations in making disability determinations and rendering rehabilitation services.

In addition, "constant pressure from government, administrators and patients seeking disability certifications would cause increasing harassment of the . . . profession."

A SPECIAL BOOKLET entitled "Federal Income Tax Liability of Physicians" is now available from the AMA's Law Department, 535 North Dearborn Street, Chicago 10. The booklet should be of much assistance in filing income tax returns.

Know Your Congressmen

VIRGINIA CONGRESSIONAL DISTRICTS, 84TH CONGRESS

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A. Willis Robertson (D) ..... Lexington

Washington Address: Senate Office Building

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For Better Government . . .

Get the Facts

Study the Issues

Form Your Opinion

Inform Your Congressmen

1. He should be addressed as Representative John Doe or Senator John Doe—not Mister.
2. Be brief, but not terse.
3. Be specific, positive—don't hedge.
4. Give him the local viewpoint—how the national issue would affect community health, your practice, your patients.
5. Letters should be dignified.
6. And reasonable—don't ask the impossible.
7. But request action—your Congressman was elected to *do* something.
8. Make it *your* letter—on your letterhead, in *your* style.
9. Request an answer—you've told him where *you* stand, now ask him where *he* stands.
10. Be appreciative—thank him for good things he does.

Personalities....

Big Chief
Williams,
His Son
and
Dr. Thompson

A
Likely
Story!

Dr. Ball
Dr. Fisher
Mrs. Ball

Dr.
Lynch
and
His
Harem

The Kings and their Queens

Sunny Side Up

Mrs. Lowe
Dr. Farmer
Att. Gen. Almond

Misery Loves Company—

Mrs. Flanders and Dr. and Mrs. Emlaw

Four Horsemen—

Drs. Hancock, Bailey, Hamner and Hagood

You Name 'Em

First Award

Drs. Bosher and Decker

President's Message....

Corporate Practice of Medicine

FOR many years the practice of medicine by corporations has been unlawful in Virginia and all other states. In its search for more revenue, some hospitals have turned to exploitation of physicians in violation of this statute. Thus far radiologists, pathologists, anesthesiologists and physiatrists have borne the brunt of the attack.

Tensions between lay hospital management and physicians have increased all over the nation. Recently, the Iowa Hospital Association entered litigation against its State Board of Medical Examiners and the Iowa Association of Pathologists, challenging the specialty of pathology as a branch of medicine. The Attorney-General of Virginia adds further confusion through his interpretation excluding radiology from the practice of medicine under the Virginia Medical Practice Act.

The position of the hospital specialist as a practitioner of medicine has been clearly defined by the House of Delegates of the American Medical Association. "The practice of anesthesiology, pathology, physical medicine and radiology are an integral part of the practice of medicine, in the same category as the practice of surgery, internal medicine or any other designated field of medicine."

If hospital administrators are successful in their efforts to alter the law to permit corporate practice of medicine, it is probable that surgeons, internists, pediatricians and other specialists will eventually be hired by the hospital and their services sold at a profit. A battery of general practitioners attached to each hospital would then complete the picture of hospital domination of the medical profession.

It is thus obvious that a proper solution to the problem of the hospital-physician relationship is important to every practitioner of medicine.

As is true in all controversies, there is a need for clear, objective thinking among those who must meet at the conference table to evolve the necessary solution. Then, only in an atmosphere of mutual confidence may these differences be settled.

James P. King

President

Editorial . . .

Our New Seal

IN THE Fall of 1954, a committee was appointed by Dr. Carrington Williams to bring in recommendations for a seal for The Medical Society of Virginia.

This was not the first reference to a seal in our Society. The minutes of November 12, 1873, quoted Dr. F. D. Cunningham, corresponding secretary, as stating that "he had a seal of the former State Medical Society, and as this Society has not yet adopted any special design for one—though the Constitution calls for a seal—he would place this at the disposal of the Society." This seal had come into the possession of Dr. Cunningham's father when the elder Cunningham was President of the State Medical Society in 1847 and had been laid aside when the earlier organization was discontinued in 1858. All efforts to locate this seal have been unsuccessful.

A cover of the Virginia Medical Monthly published in 1874 showed a small design consisting of a snake entwined about a sun-dial or garden ornament but this appeared to be a filler supplied by the printer rather than an official seal.

At the Richmond meeting of The Medical Society of Virginia last October about a dozen designs were submitted to the Council. These were chiefly adaptations of state and other seals of local interest. By a happy coincidence, a member of the committee had received recently a Greek silver coin minted in Cos during the first century B. C. depicting Aesculapius. This was found to be the earliest known portrayal of this Greek physician. Designs incorporating the head of Aesculapius and also the reverse of the coin, which showed him as a seated figure with a highly individualized serpent, were also submitted. The design showing only the head had the virtue of simplicity. This was recommended by the Council and adopted by the House of Delegates. This seal appears for the first time on the cover of this issue of the Journal.

Aesculapius (or Asklepios) was the Greek God of Medicine. He is said to have

been the son of Apollo and the nymph Coronis. Cheiron, the centaur, taught him the art of healing. His sons, Machaon and Podaleirius, are mentioned in the Iliad as serving in the Greek Army during the Trojan War. More famous was their sister Hygieia, Goddess of Health, whose name continues as a household word to the present time.

Temples were erected to Aesculapius throughout Greece. Case records of those cured by him have been preserved in the form of votive tablets from the fourth century B. C. in his shrine at Epidaurius. His success, in fact, led to his untimely demise, for Zeus, fearful that under his ministrations all men would become immortal, slew him with a thunderbolt.

The design chosen for your Society, therefore, recalls medicine's proud history and it is hoped that we today may prove worthy of our seal and of our heritage.

Society Proceedings . . . .

Richmond Academy of Medicine.

The Eye, Ear, Nose and Throat Society of the Academy held its meeting on November 22nd. Dr. Philip Knapp, Assistant Professor of Opthalmology Institute of Opthalmology, College of Physicians and Surgeons, New York, spoke on "The Cross Eyed Child".

Southern Medical Association.

At the 49th Annual Meeting of this Association, held in Houston, November 14-17, Dr. W. Raymond McKenzie, Baltimore, was installed as president and Dr. J. P. Culpepper, Jr., Hattiesburg, Miss., was named president-elect. A special "Golden Anniversary Key" was presented to Mr. C. P. Loranz, Advisor and Professional Relations Counselor, in recognition of his 43 years of loyal and efficient service to the Association.

The 1956 meeting of the Association will be the "Golden Anniversary" and will be a conjoint meeting with the Medical Society of the District of Columbia in Washington, November 12-15.

Virginia Obstetrical and Gynecological Society.

At the annual meeting held in Richmond on October 18th, Dr. George S. Hurt, Roanoke, was made president; Dr. James M. Habel, Jr., Suffolk, vice-president; president-elect Dr. Millard B. Savage, Norfolk; and secretary-treasurer, Dr. Chester D. Bradley, Newport News.

News . . . .

Calendar of Coming Events

ANNUAL CONGRESS ON INDUSTRIAL HEALTH—Sheraton-Cadillac Hotel, Detroit, Michigan—January 23-24.

MEDICAL SOCIETY EXECUTIVES POSTGRADUATE SEMINAR—Drake Hotel, Chicago, Illinois—February 6-8.

ANNUAL MEETING OF THE AMERICAN COLLEGE OF RADIOLOGY—Drake Hotel, Chicago, Illinois—February 10.

52ND ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE—Palmer House, Chicago, Illinois—February 11-14

AMERICAN COLLEGE OF SURGEONS, Regional Meeting—The Bellevue-Stratford, Philadelphia, Pennsylvania—February 13-16.

INTERNATIONAL COLLEGE OF SURGEONS, Regional Meeting—Greenbrier Hotel, White Sulphur Springs, West Virginia—February 13-15.

VIRGINIA CHAPTER OF THE AMERICAN ACADEMY OF GENERAL PRACTICE—The Homestead, Hot Springs, West Virginia—March 2-3-4.

SOUTHEASTERN SURGICAL CONFERENCE—John Marshall Hotel, Richmond, Virginia—March 12-15.

5TH ANNUAL AMERICAN ACADEMY OF GENERAL PRACTICE SCIENTIFIC ASSEMBLY—Armory, Washington, D. C.—March 19-22.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY—Convention Cruise to Havana and Nassau (sailing from Norfolk)—May 26-June 2.

Committee on Arrangements.

Drs. Allen Barker and Reverdy H. Jones, Jr., are co-chairmen of the local committee on arrangements for the annual meeting of The Medical Society of Virginia in Roanoke, October 14-17. Other committees are: Scientific Exhibits, Dr. Marcellus A. Johnson, III, chairman; Technical Exhibits, Dr. Alexander McCausland, chairman, and Drs. Theron H. Hass, Harry J. Minarick and Walter A. Porter; Golf, Dr. Rufus P. Ellett, Jr., Chairman, and Drs. C. C. Hatfield and George W. McCall; Hotels and Halls, Dr. Robert S. Hutcheson, Jr., chairman, and Drs. Homer A. Sieber, Robert E. Paine, Jr., Rufus Brittain, and W. R. Whitman, Jr.; Entertainment, Dr. Hugh H. Trout, Jr., chairman, and Drs. J. T. Showalter, W. M. Moir, Charles A. Hefner, and F. Willson Daily; Press and Publicity, Dr. Hugh J. Hagan, Jr., chairman, and Drs. W. Randolph Chitwood and Charles L. Crockett, Jr.; and Ladies' Entertainment and Auxiliary, Mrs. Edwin J. Palmer, chairman.

Oldest Active Practitioner.

A recent query as to the oldest practicing physician in Virginia revealed the following information.

Dr. Sidney J. Tabor, born August 31, 1867, is in active general practice in Portsmouth.

Dr. Ramon D. Garcin, born September 19, 1867, is a practicing internist in Richmond.

Dr. Halsted S. Hedges, born September 30, 1867, is practicing opthalmology in Charlottesville.

Dr. J. C. Cutler, born November 9, 1867, is still practicing as a proctologist in Newport News.

Do you know of any doctor who is older than the four listed above—and still in active practice? If so, the Monthly would like to have this information.

Dr. Edward M. Holmes, Jr.,

Director of Public Health of the City of Richmond, has been named president-elect of the American Association of Public Health Physicians, at the meeting of this Association in Kansas City in November. By acceptance of the post, Dr. Holmes immediately becomes a member of the board of trustees of the association and will remain on the board for five years.

American Academy of General Practice.

The Eighth Annual meeting of the Academy will be held in Washington, D. C., March 19-22. There will be twenty-six outstanding speakers discussing subjects ranging from cardiac emergencies to primary wound repair. There will also be more than sixty scientific and two hundred and fifty technical exhibits. The program also includes two live clinics, a symposium on obstetrics, and special tours through the National Institutes of Health at Bethesda.

Dr. Douglas Vanderhoof,

Richmond, has been elected honorary senior warden for life at St. Stephen's Episcopal Church. This is the first time such an honor has been conferred by this church.

State Health Department News

Dr. R. H. Laning retired on December 1 after eight years' service as Director of the Amherst-Nelson Health District.

Dr. Patricia Hunt, having completed three month's orientation given new medical personnel, will assume the duties of Acting Director of the Amherst-Nelson Health District.

Dr. J. K. Farish, Jr., has also completed the usual indoctrination, and has assumed duties as Acting Director of the Fredericksburg-King George-Spotsylvania-Stafford Health District, succeeding Dr. H. D. Crow, who was transferred to the Tri-County Suffolk District.

Dr. Henry C. Evans, having completed his orientation, has assumed duties as Assistant to Dr. Leroy D. Soper in the recently combined Halifax-Pittsylvania-Danville Health District.

T. Duckett Jones Memorial.

Dr. Paul D. Camp, Richmond, has been named chairman of a state committee and a member of a national committee to raise $150,000 for a memorial fund to honor the late Dr. T. Duckett Jones. Income from the fund will support outstanding young scientists seeking investigative careers in rheumatic fever and related fields. Dr. Jones was a native of Petersburg, a son of the late Dr. Bolling Jones, a graduate of V.M.I. and the University of Virginia Medical School. He was considered an internationally famous authority on rheumatic heart disease.

Annual Winter Clinic.

The Louise Obici Memorial Hospital, Suffolk, will hold its annual winter clinic on January 18th. The morning program is on The Present Status in the Management of Coronary Heart Disease and the speakers are Dr. Paul Camp, Richmond; Dr. Eugene A. Stead, Professor of Medicine, Duke University; Dr. John A. Boone, Professor of Medicine, Medical College of South Carolina. In the afternoon the subject will be Medical and Surgical Diseases of the Chest. Dr. Sol Katz, Chief of the Pulmonary Disease Division of the District of Columbia General Hospital, Georgetown and George Washington University Schools of Medicine; Dr. Edgar Davis, Professor of Thoracic Surgery, Georgetown University Medical Center; and Dr. Philip Hodes, Professor of Radiology, University of Pennsylvania, will be the speakers.

The Atlanta Graduate Medical Assembly,

Which will be held in Atlanta, February 20-22, promises another excellent speaking faculty and a streamlined and sharpened agenda to incorporate features which have attracted most favorable comment from the doctors who have been attending each year. The speaking faculty will consist of Drs. Philip K. Bondy, Woodbridge, Conn.; Charles C. Harrold, New York; Theodore Winship, Washington, D. C.; Russell L. Dicks, Durham, N. C.; Willis J. Potts, Oak Park, Ill.; Clyde L. Randall, Buffalo, N. Y.; Alexander D. Langmuir, Atlanta; Jack D. Myers, Pittsburgh; Charles H. Hendricks, Cleveland; Ralph B. Cloward, Honolulu, T. H.; Arnall Patz, Baltimore, Ivan L. Bennett, Jr., Baltimore; Meredith F. Campbell, Miami; Philip Thorek, Chicago; Samuel Kaplan, Cincinnati; Wilburt C. Davison, Durham, N. C.; George Saslow, Newton Highland, Mass.; Fred J. Hodges, Ann Arbor, Mich.; and C. Walter Lillehei, Minneapolis, Minn.

Further information may be obtained from the Assembly at 15 Peachtree Place, N. W., Atlanta, Ga.

Dr. J. A. B. Lowry,

Crewe, was doubly honored recently when he

received the second annual Lions Club plaque for good citizenship and he was paid tribute in a program patterned after the television show "This is Your Life". This was participated in by old friends, men he has been associated with in various projects and members of his family.

Fellows of American College of Surgeons.

At the recent meeting of the College in Chicago, fourteen Virginians were made Fellows. They are: Drs. Milton J. Hoover, Jr., Martin Markowitz, and William McC. Eagles, Richmond; Forrest M. Swisher, Arlington; Drake Pritchett, Danville; Wilbur J. Baggs, Jr., and William H. Woodson, Newport News; William E. Byrd and John H. Hill, Norfolk; Charles B. Bray, Jr., and William P. Tice, Roanoke; S. Randolph Penn, Waynesboro; and Lester A. Wilson and M. K. Humphries, Jr., Charlottesville.

National Association of Medical Assistants.

Mrs. Lucy Carwile and Mrs. Doris Paris, Medical Office Personnel Association in Lynchburg, are shown with Dr. John Wyatt Davis, Jr., just before they left for Kansas City, Kansas, on November 4th to attend a two day meeting to create a national association of medical assistants. There were 74 medical assistants registered, representing 17 states. The name of the organization is to be the American Association of Medical Assistants, and the 1956 meeting will be held in Milwaukee. Mrs. Paris will be the Virginia representative and a member of the organizational committee.

Dr. Guy C. Richardson

Bristol, has been honored with the President's Citation for his work in the National Employ the Physically Handicapped movement. This was presented to him for his service in providing medical care and occupational opportunity for the physically handicapped.

Medical and Surgical Symposium.

The Thirteenth Annual Watts Hospital Medical and Surgical Symposium will be held in Durham, N. C., February 8th and 9th. The symposium will be held at the Washington Duke Hotel and speakers will include Drs. Louis A. Krause, Baltimore; James W. Carpenter, Chicago; Harry S. N. Greene, New Haven, Conn.; Stanley O. Hoerr, Cleveland; Albert W. Diddle, Knoxville; S. Gordon Castigliano, Philadelphia; Robert W. Johnson, Baltimore; William B. Castle, Boston; Monroe J. Romansky, Washington. On the 8th there will be a panel discussion on "Recent Advancements in Management of Malignant Disease" and on the 9th there will be clinics at Watts and McPherson Hospitals.

Our readers are cordially invited to attend this Symposium and further information may be obtained from Dr. James E. Davis, 1200 Broad St., Durham, N. C.

American College of Surgeons.

A Sectional Meeting of the College will be held in Philadelphia on February 13-16. This meeting may be attended by any doctor interested. Non-fellows pay a five dollar registration fee, but interns and residents pay no fee. Headquarters are the Bellevue-Stratford Hotel.

Dr. Houston L. Bell,

Roanoke, has recently returned from New York City where he took a special course in rhinoplasty given by Dr. Samuel Fomon.

Medical Officer Needed.

The Federal Civil Defense Administration, Olney, Md., is in need of a full time Medical Officer GS-12 at $10,065.00 per annum. It is required by the Civil Service Commission that this individual have a Doctor of Medicine degree and a considerable background in Medical Administrative work. He would be located at Olney, Md., and act in the capacity of Advisor to Civil Defense organizations in the States of Maryland, Delaware, Ohio, Kentucky, Virginia, West Virginia, Pennsylvania and the District of Columbia in health problems related to Civil Defense.

The International Academy of Proctology

Announces its annual cash prize and certificate of merit award contest for 1955-1956. The best unpublished contribution on proctology or allied

subjects will be awarded $100.00 and a certificate of merit. The competition is open to all physicians in all countries whether or not affiliated with the Academy. Entries are limited to 5,000 words, must be typewritten in English, and submitted in five copies. All entries must be received no later than the first of February, 1956, and should be addressed to the Academy at 147-41 Sanford Ave., Flushing, N. Y.

Virginia Nephrosis Foundation

On November 17, 1955, a group of interested people gathered at First Baptist Church, Richmond, in an attempt to form a Virginia Nephrosis Foundation. Forty to fifty people attended this first meeting and decisions made at that time were as follows:

1. To form a Virginia Nephrosis Foundation.
2. To organize a temporary executive board consisting of
   a. President, Mr. E. S. Hirschler
   b. Vice-President, Mr. Wallace E. Parham
   c. Secretary-Treasurer, Mr. L. A. Blackburn
   d. Medical Advisory Board, Edwin L. Kendig, Jr., M.D. and Thomas P. Overton, M.D.
3. To inform the people of Virginia of such a foundation.
4. To stimulate and secure membership.

The ultimate goal of this foundation is one of better care, study and treatment of nephrosis in Virginia.

Those people who might be interested, please contact Mr. L. A. Blackburn, Richmond Federal Building, Richmond.

International College of Surgeons.

There will be a sectional meeting of the College at the Greenbrier Hotel, White Sulphur Springs, from February 12th to 15th. The profession is cordially invited to attend.

For Sale.

Hospital-Clinic. Yes, it is most unusual to find one on the market. But due to circumstances this one must be sold. Here is a grand opportunity for two general practitioners not only to make a lot of money but to render a great service to this community. This hospital has 10 rooms, plus very modern living quarters, brick construction, with beautifully landscaped 2 acre tract, and just about every type equipment you would need, which is practically new. It is available at a sacrifice of $85,000.00. Financing can be arranged. Contact realtors, L. T. Jonas and J. Hunter Roberts, associated with the firm of Jno H. Windel, Inc., 34 W. Kirk Ave., Roanoke, Va., phone 3-2478. (*Adv.*)

For Rent.

Doctor's office, three doors west of Lee Medical Building, Richmond. Will remodel to suit doctor. Write "Office", care the Monthly, Box 5085, Richmond 20. (*Adv.*)

For Sale.

Suburban properties. Homes and timber lands, located in the vicinity of Richmond and Hopewell. Sound investment opportunities. Call or write W. L. Broaddus, phone 844, P. O. Box 479, Hopewell, Va. (*Adv.*)

For Sale.

Complete physicians office equipment, including GE vertical fluoroscope, Cambridge direct writing electrocardiograph, etc. Write "Equipment", care the Monthly, P. O. Box 5085, Richmond 20. (*Adv.*)

Desires Location.

General surgeon desires association or solo practice in city with available hospital facilities. Board eligible, 34, Category IV, university trained including sub-specialties. Available July 1956. Write #55, care the Monthly, P. O. Box 5085, Richmond 20. (*Adv.*)

Obituaries . . . .

Dr. David Alexander Christian,

Appomattox, died on November 18th, having been in ill health for many months. He was seventy-five years of age and graduated from the Medical College of Virginia in 1908. Dr. Christian was one of the county's oldest physicians and five years ago, the townspeople and medical colleagues unveiled a portrait of him at the courthouse. He had been a member of The Medical Society of Virginia for forty-four years. Five daughters and a son survive him.

Dr. Moncure.

In publishing notices of the death of Dr. P. St. L. Moncure, Norfolk, in the November and December issues of the journal, the Monthly failed to state that Mrs. Moncure survived her husband. We regret that this error was made but we had only the information obtained from the newspapers.

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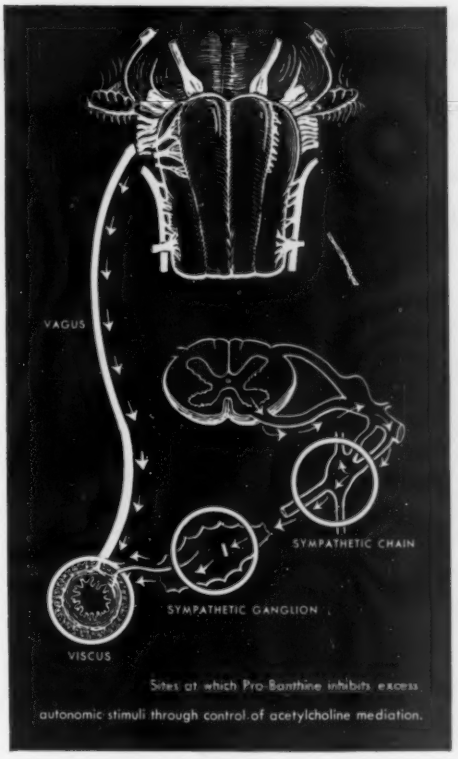

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1. Schwartz I. R.; Lehman, E.; Ostrove, R., and Seibel, J. M.: Gastroenterology *25*:416 (Nov.) 1953.
2. Roback, R. A., and Beal, J. M.: Gastroenterology *25*:24 (Sept.) 1953.

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John E. Magielski, M.D. — Ann Harbor, Mich.
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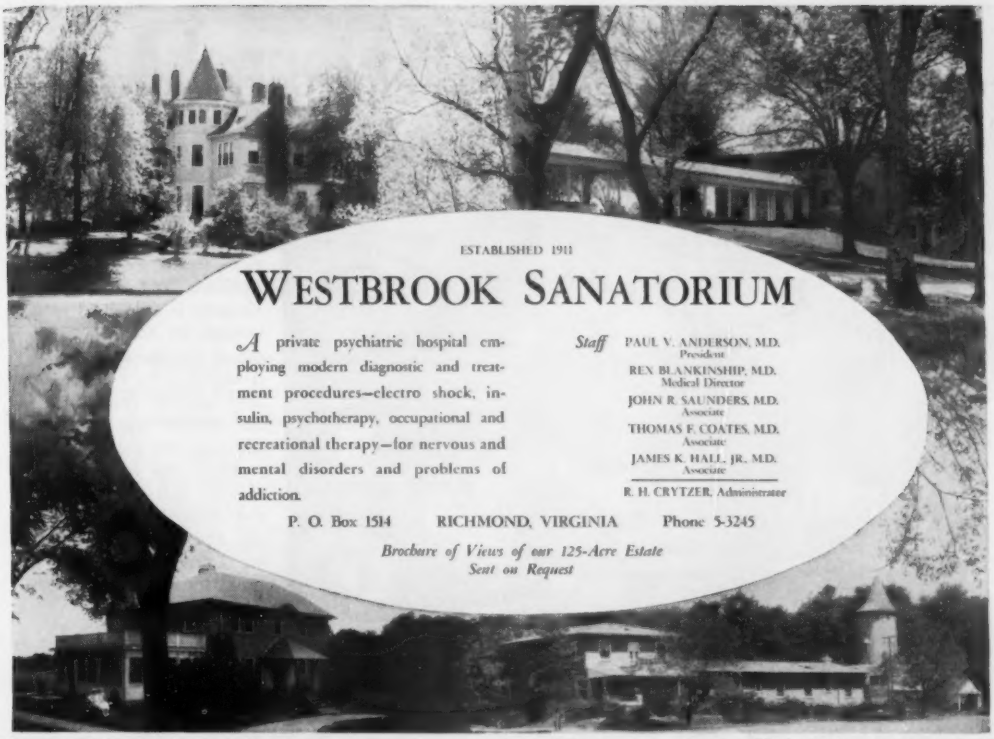

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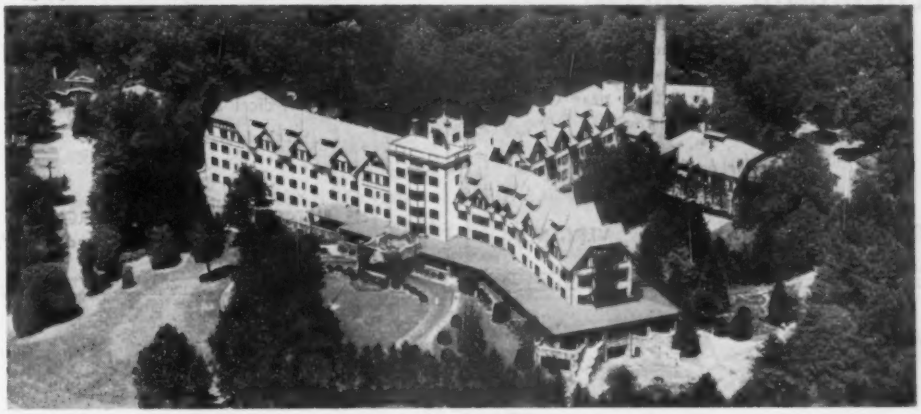

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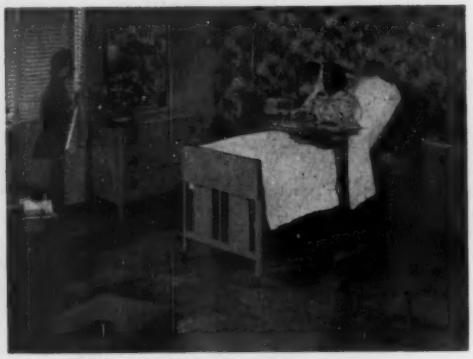

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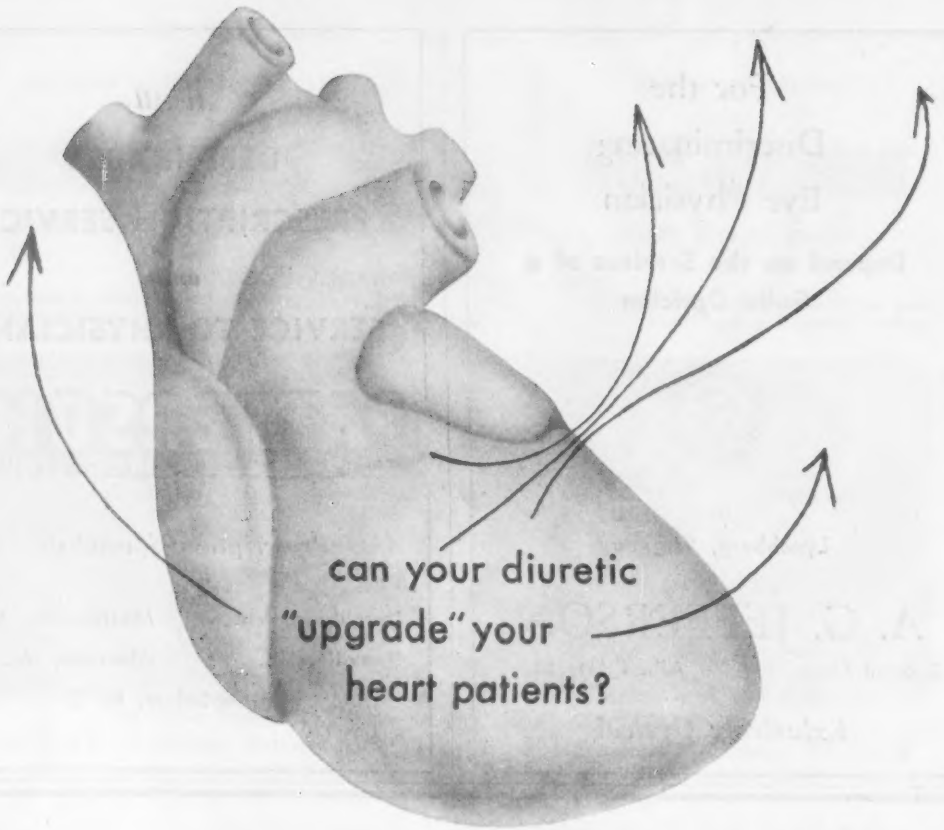

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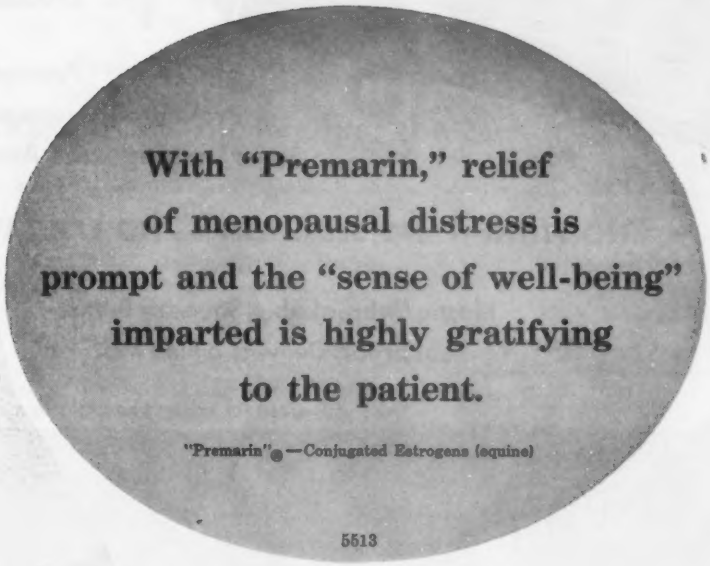
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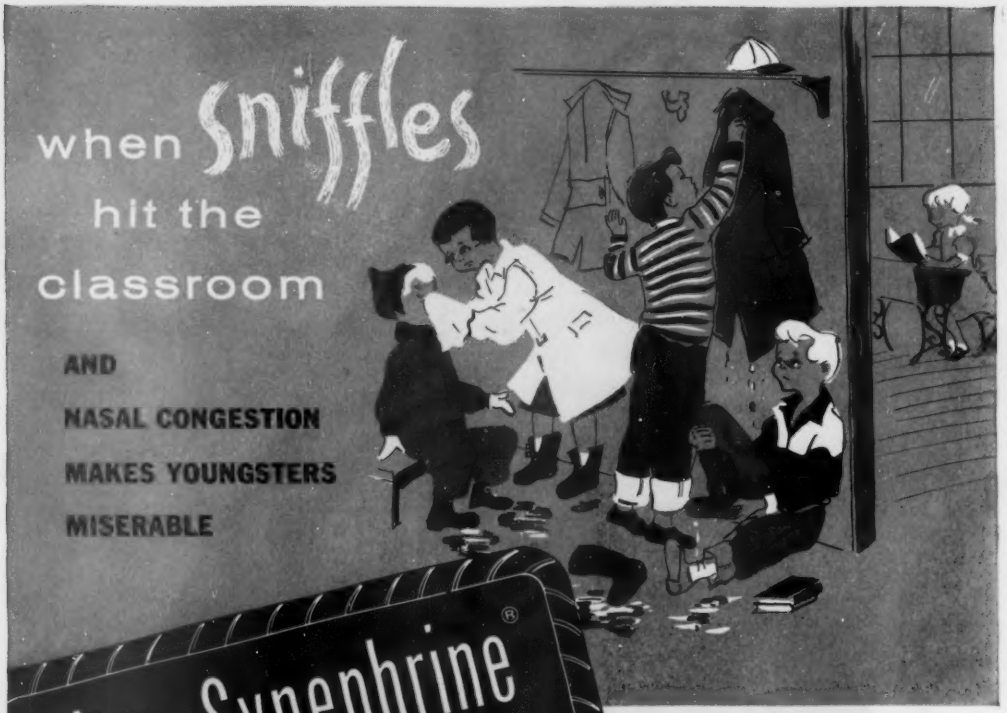

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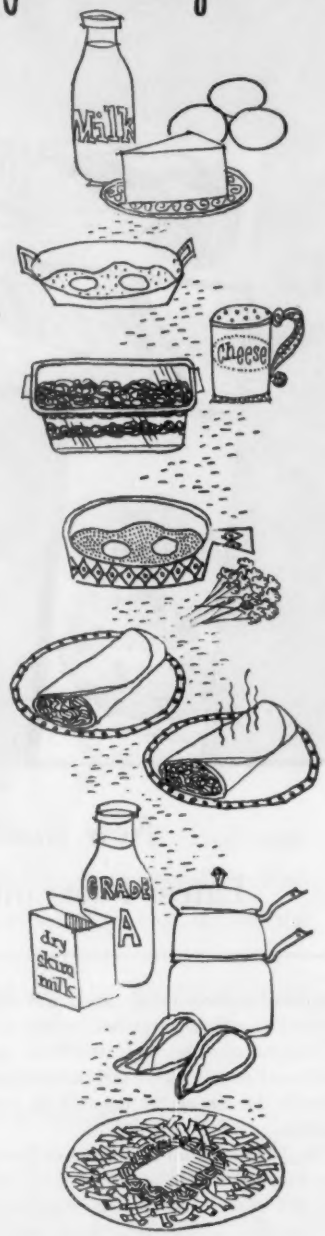

In these, the trio plays a solo—

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1. Pomeranze, J. et al.: Angiology, June, 1955.
2. Freedman, L.: Angiology 6:52, Feb. 1955.

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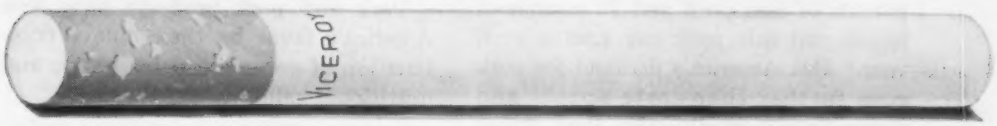

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TABLE I	Protein Gm.	Thiamine mg.	Niacin mg.	Riboflavin mg.	Iron mg.	Phosphorus mg.	Calories
Pork Chops, without bone, cooked, 3 oz.[2]	20	0.71	4.3	0.20	2.6	200	284
Ham, without bone, cooked, 3 oz.[2]	20	0.45	4.0	0.20	2.6	202	338
Pork Sausage, cooked, 3 oz.[3]	14	0.42	2.8	0.20	2.1	139	396

3.5 ounces of fresh pork loin, equivalent to approximately 3 ounces of cooked loin, contains 0.47 mg. pantothenic acid;[4] 0.10 mg. pyridoxine;[4] 0.005 mg. biotin;[5] 36 mg. inositol;[4] 0.08 mg. folic acid;[4] 0.0027 mg. vitamin B_{12};[6] 63 mg. chlorine;[7] 0.1 mg. copper;[7] 20 mg magnesium;[7] 280 mg. potassium;[7] 70 mg. sodium;[7] and 0.01 mg. manganese.[7]

TABLE II — Nutrients and Calories of Cooked Pork Chops (3 ounces) Expressed as Percentages of Recommended Daily Dietary Allowances[8]

Percentages of Allowances for:	Protein	Thiamine	Niacin	Riboflavin	Iron	Phosphorus	Calories
Girls, 13-15 years of age; weight, 108 lb.; height, 63 inches.	25%	55%	33%	10%	17%	15%	11%
Women, 25 years of age: weight, 121 lb.; height, 62 inches.	31%	59%	36%	14%	22%	17%	12%
Pregnant Women (3rd trimester)	25%	47%	29%	10%	17%	13%	11%

The nutritional statements made in this advertisement have been reviewed by the Council on Foods and Nutrition of the American Medical Association and found consistent with current authoritative medical opinion.

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